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THE BACTERIOLOGY OF SPREADING PERITONITIS COMPLICATING ACUTE PERFORATIVE APPENDICITIS

A CLINICAL AND EXPERIMENTAL STUDY

JOHN O. BOWER, M.D., JOHN C. BURNS, M.D., AND HAROLD A. MENGLE, M.D., PHILADELPHIA, PA.

(From the Department of Research Surgery, Temple University School of Medicine, Philadelphia General Hospital)

FOR over 15 years the senior author has been investigating acute appendicitis and spreading peritonitis following perforative appendicitis. From 1928 to 1933 inclusive, annual surveys of the appendicitis mortality in the city of Philadelphia were made and another survey is now under way for the years 1934-36.

This clinical investigation has resulted in the development of a prophylactic campaign for the reduction of the mortality of acute appendicitis which has been successfully carried out in Philadelphia through the Department of Public Health and which is now being conducted throughout Pennsylvania and New Orleans, Cincinnati, and other large cities in the United States, following the Philadelphia plan.

The mortality of acute appendicitis in Philadelphia during the years 1929 to 1933 inclusive was reduced from 5.95 to 3.54 per cent. A study of the factors contributing to this reduction shows that the earlier hospitalization of patients and the less frequent administration of laxatives before admission were responsible. During this same period of time, the mortality of spreading peritonitis, the cause of 81.5 per cent of the deaths attributed to acute appendicitis, was not materially reduced. In 1930 it was 26 per cent; in 1931, 24 per cent; in 1932, 22 per cent; and in 1933, 24 per cent. Because of this persistently high mortality, the investigations herewith reported were begun.

A study of the bacteriology of spreading peritonitis in an effort to establish the identity of the causative organisms seemed indicated, especially in view of the conflicting reports in the literature regarding the rôle played by the anaerobes. As our investigations proceeded, we

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found that our results agreed with the majority of other investigators so far as the aerobic flora are concerned. The significance of the anaerobic flora, notably *Cl. welchii*, mentioned in other bacteriologic studies of peritonitis, has remained an uncertain quantity, due to the lack of unanimity among reporters, both as to their incidence and etiologic significance. A review of the literature will reveal this conflict of opinions.

REVIEW OF THE LITERATURE

Laruelle,[1] in 1899, was the first to call attention to and to emphasize the importance of *B. coli* in peritonitis, secondary to intestinal lesions. He found it in pure culture in two cases of perforated appendix and in the peritoneal cavity in the absence of perforation. Fraenkel,[2] in 1890, discovered the streptococcus; Jalaguier,[3] in 1892, contributed *Staph. albus.* In 1893 Tavel and Lanz[4] distinguished three different forms of streptococci occurring in the bacterial flora of peritonitis. They did not identify anaerobes. Barbacci[5] discovered that pneumococcus was present in spreading peritonitis. Welch and Flexner,[6] in 1896, reported the presence of *B. aerogenes capsulatus* in peritonitis. Tavel and Lanz,[7] in 1898, pointed out that obligate anaerobes appeared in the exudate of peritonitis, but they were unable to culture them. In 1899 Veillon and Zuber[8] were able to grow obligate anaerobes in pure culture from peritoneal exudate. They reported the finding of anaerobes in 98 per cent of the cases of perityphlitis. *B. ramosus* and other putrefactive anaerobes were reported; *Cl. welchii* was mentioned as *B. perfringens.* Their report was based on 26 cases of appendicitis; 14 were complicated by peritonitis; 10 had free peritoneal exudate or local abscess with gangrene of the appendix; 2 had pure empyema of the appendix. In addition to the anaerobes, they reported *B. coli* and *Str. pyogenes.* They found *D. lanceolatus, B. proteus vulgaris* and pneumococcus in the peritoneal exudate. In 1896 Welch and Flexner reported 7 cases of peritonitis in which *B. aerogenes capsulatus* was found; and in 1900 Welch[9] reported 13 cases of diffuse peritonitis, in the exudate of which *B. aerogenes capsulatus* was found. Ten of the cases had perforations of the intestinal tract. Of these, 4 were typhoid ulcers; 4, gastric ulcers; 1, strangulated gangrenous intestine; and 1, a cancerous ulcer of the duodenum. In addition he said: "We have found the gas bacillus *(Cl. welchii)* mixed with the other bacteria twice in circumscribed gas producing, intraperitoneal abscess resulting from perforation of the appendix veriformis." Welch also mentioned cases studied by Flexner and himself in which pneumoperitonitis caused by *Cl. welchii* occurred without perforation of the intestine.

Krogius,[10] in 1901, studied 36 cases of peritonitis of appendical origin and confirmed the work of Veillon and Zuber. In 1904 Lanz and Tavel[11] published a bacteriologic study of the contents of the normal

and the pathologic appendix and the appendical abscess in a series of 146 cases. In addition to the usual anaerobes, they obtained the vibrion septique of Pasteur. In 1904 Hewetson[12] was unable to find anaerobes in appendicitis and in only one instance in peritonitis associated with appendicitis. Dudgeon and Sargent,[13] in 1905, "failed to find any evidence to show that anaerobic organisms are of importance in appendicitis." Manahan,[14] also in 1905, in reporting the bacteriology of spreading peritonitis in 110 cases, 24 of which were due to a perforated appendix without abscess formation, found *Cl. welchii* in only 1 case. Heyde,[15] in reporting his investigations covering a period from 1908 to 1911, found *Cl. welchii* in peritonitis in 24 per cent, anaerobic streptococcus in 26 per cent, and vibrion septique in 20 per cent, in addition to the usual aerobes. His report includes 27 cases of spreading peritonitis. Runeberg,[16] in the same year, reported finding *Cl. welchii* in 11 per cent, anaerobic streptococcus in 66.6 per cent, and various other aerobes in 9 cases of spreading peritonitis associated with appendicitis. Vick,[17] in 1912, found *Cl. welchii* in 36 per cent of cases of gangrenous appendicitis and peritonitis. Ante-mortem blood cultures were negative. Post-mortem blood cultures were made in all cases in which an examination had been made before death, and in each case, with but one exception, the microorganism isolated from the blood at post mortem corresponded in all its morphologic characteristics and its biochemical reactions to that which was predominant in the local infection before death. *B. coli* was found in 82 per cent; alone in 30 per cent. Streptococci were found in 36 per cent; alone in 8 per cent; *B. coli* with *Cl. welchii,* in 22 per cent; staphylococcus with *Cl. welchii,* in 16 per cent; *Cl. welchii* and streptococcus, in 9 per cent.

In 1912 Fishbein[18] reported post-mortem bacteriologic studies of peritonitis and mentioned that the most commonly found microorganisms are *B. coli,* staphylococcus, and streptococcus. He did not mention anaerobic studies.

In 1913 Deaver[19] mentioned Heyde's work but did not corroborate his findings. He was of the same opinion as Hewestón—that aerobes play a small part in the etiology of appendicitis. The first mention of anaerobes in a textbook of surgery was made by Kelly and Noble[21] in 1914, *B. aerogenes capsulatus* being mentioned specifically. Again in 1921, in his monograph on "Peritonitis," Deaver failed to mention the anaerobes. In 1920 Ashurst[22] did not mention them. Likewise, in the 1931 edition they were ignored. Murphy[23] did not mention the anaerobes.

In 1925, Sherrill,[24] in his monograph "Peritonitis," did not include the anaerobes but mentioned that "*B. coli* may show a gas-producing property and this may excite the suspicion of the presence of *Cl. welchii.*"

Royster,[25] in 1927, in his textbook on appendicitis, did not mention *Cl. welchii* but stated that "anaerobic organisms are supposed to be productive of gangrene of the appendix and are responsible for the high-grade toxemia developed from such pathology. Without doubt, these bacteria are more often present than is usually believed."

In 1928 Weinberg, Pierot, Davesne, and Renard[26] instituted a thoroughly painstaking investigation into the bacteriology of appendicitis and associated peritonitis. The results of their investigations warrant reporting in detail. Of the aerobes, they reported finding *B. coli* in 87 per cent; enterococcus in 30 per cent; staphylococcus in 9 per cent; *B. proteus* in 9 per cent; *B. subtilis* in 9 per cent; *B. mesentericus* in 9 per cent; *B. lactis aerogenes* and *B. faecalis alcaligenes* each, 3 per cent; *B. tetragenes, B. pyocyaneus, B. d.morgan* each, 1 to 2 per cent.

The anaerobes most frequently found were *Cl. welchii, B. histolyticus, V. septique, B. sporogenes, B. oedematiens, B. ramosus,* and anaerobic cocci (9 different specimens averaging 18 per cent). These authors write: "We have shown that all the anaerobes of gas gangrene may take part in the flora of acute appendicitis and have isolated some particular specimens such as *Bacillus de Ukil.* It is undeniable that among the aerobic microbes the primary rôle is played by the *Bacillus coli,* not because of its great frequency in the flora of the infection, but because of the nearly constant pathogenic power of all the strains of this species isolated. As for the anaerobic microbes, it is certain that the most important rôle is played by the *Clostridium welchii (Bacillus perfringens),* of which all strains were very pathogenic. The appendicitides caused by other species of gas gangrene—*Vibrion septique, Bacillus histolyticus,* and *Bacillus sporogenes*—were especially grave."

Their investigations were based on the study of 100 cases of gangrenous appendicitis or fulminating appendicitis. They observed that *Cl. welchii* increased in frequency with the severity of the process. While it was identified in only 35 per cent of all the appendices examined, they found it in 29 of the 58 specimens, or in 58 per cent, of gangrenous appendicitis. *B. ramosus* was isolated from 14 per cent of the cases; *V. septique* and *B. sporogenes* were found only in the gangrenous form.

They also found that the anaerobes increased in frequency as the number of aerobes increased in the flora, and the more complex the flora, the greater the number of anaerobic species. "While in the appendicitides with 2 microbes, one finds 25 anaerobic species to 75 aerobic species, in those of 3 microbes the number of anaerobes is increased to 59 to 62 aerobes; when 4 microbes were found there were 58 anaerobes to 44 aerobes; when 5, 49 anaerobes to 31 aerobes; when 6, 14 to 10; when 7, 9 to 5." To quote further: "Whatever the order of entry of the anaerobic microbes during the course of acute appendicitis, the important fact for us is that these microbes indirectly

play a rôle of first importance in the genesis of the disease. The pathogenicity of all our strains has been verified experimentally on animals and, moreover, their infectious power and identification has been established by neutralization with specific sera."

Weinberg quotes Hines as having found the *B. welchii* in 95 per cent of human feces.

In 1931[20] Meleney, Harvey, and Zaytseff-Jern reported a correlation of the bacteriology of the peritoneal exudate and the clinical course of the disease in 106 cases of peritonitis. These authors have in this contribution not only added to our knowledge of the bacteriology of the disease, but also have in an excellent manner correlated these findings with its clinical manifestations. The material examined bacteriologically was that "actually present in the peritoneal cavity and presumably left behind, to some extent, at least after operation . . . rather than . . . the contents or the wall of an organ that had been removed from that patient and was no longer a factor in his illness. We therefore centered our attention almost exclusively on the peritoneal exudate."

These authors reported 25 cases of known perforation of the appendix. Two of these had an acute local peritonitis; 12, a local peritonitis; and 11, an acute diffuse peritonitis.

All the smears from the fluid taken from the spreading cases showed at least 2 forms of bacteria; and 4 of the smears showed 3 forms. No cultures were sterile and smears of the 11 yielded from 3 to 4 different species. *B. coli* nonhemolytic was found in every case, and in 4 the hemolytic variety as well. More than one-half showed *Str. viridans,* and anaerobically *Cl. welchii* was found in 25 per cent, while 33⅓ per cent of the cases yielded an anaerobic streptococcus.

In a subsequent contribution in October, 1932, these authors[27] studied the synergism of bacteria present in peritoneal exudate. They concluded there was a definite synergistic action producing in animals a lethal infection of the peritoneum.

These writers feel, however, that "*Cl. welchii* is not particularly virulent in the peritoneal cavity and that it is essentially saprophytic rather than pathogenic." Inasmuch as their conclusions were based on experimental work carried out on white mice, which have a resistance to *Cl. welchii* almost equal to that of the rat, this statement leaves room for some doubt.

Recent editions, such as Homan's *Practice of Surgery,* 1933,[28] do not mention the anaerobes as factors in the etiology of peritonitis. DaCosta, in 1931,[29] stated "gas-forming bacilli cause a form of peritonitis in which gas forms in the abdominal cavity." Romanis and Michiner, in 1932,[30] wrote that "of the aerobes, the most common are Bacillus coli, Streptococci less frequently, Staphylococcus, Pneumococcus and Gonococcus. Of the anaerobes, *Clostridium welchii* very frequently.

. . . It would appear that much of the toxemia of appendicitis is due to this organism . . . excellent results have been obtained in some cases by the use of *C. welchii* serum."

Boyd's *Surgical Pathology*, 1933[31] under "Peritonitis," mentions as an etiologic factor "certain anaerobic bacilli." Lewis's *Practice of Surgery*, 1933, revised to date, gives an historical review, mentions *Cl. welchii*, and quotes Weinberg extensively. On the other hand, Nelson's *Loose-Leaf Surgery*, 1933,[32] also revised to date, does not mention the anaerobes.

THE BACTERIOLOGY OF SPREADING PERITONITIS IN MAN AND OF LAXATIVE INDUCED SPREADING PERITONITIS IN THE DOG

Material and Technique.—In man the material used was obtained, without exception, at operation; in the dog, at operation and autopsy. Methods of obtaining the material, its preparation and study were the same in both man and dog. The material taken from the human being, however, could be obtained from only that part of the process which was exposed at operation. In approximately 38 per cent of the cases a small transverse incision was made. Moreover, the central zone of infection only was exposed; no attempt was made to remove material from the middle and peripheral zones. Frequently the appendix was not removed; many times it was not seen. When the exudate was profuse or moderate in amount, it was removed by aspiration using a small glass syringe; if small in amount, cotton swabs were used. It was not possible to send all specimens to the laboratory immediately because of the time of operation, etc. Those specimens that were held were placed on ice and transferred to the laboratory at the earliest possible time. Both smears and cultures were made. The blood of the heart was also cultured in the cases of human beings that came to autopsy; similar cultures were made in dogs to check on the antemortem invasion of the general tissues of the body by bacteria. Smears were made in every case and stained by Gram's method, and an attempt made to identify the organisms present morphologically before culture.

Method Used for Identifying Aerobic Microorganisms.—The material used for the aerobic cultures, in the majority of instances, was placed directly on a blood agar plate; but where the material was small in amount or if for other reasons there was doubt of obtaining a growth, the material was cultured in bouillon and then transferred to the blood agar plate after twenty-four hours. The aerobic organisms were recognized by their colony formation on the blood agar plate and by their morphology.

An effort was made to obtain pure cultures by using a hand lens, picking out individual colonies with a fine platinum loop and reinocu-

lating them upon glucose bouillon. The original plates were incubated for seventy-two hours, if necessary, to insure against missing any slowly growing organisms. After definite growth was obtained, either in the original or subcultures, smears were again made, stained by Gram's method, and the organisms identified. In cases of doubt the carbohydrate fermentation reactions were studied. By this method identity was established for *B. coli, Str. viridans, Str. non-haemolyticus, Str. faecalis, Staph. albus, B. proteus* and diphtheroids.

Method Used for Identifying Anaerobic Microorganisms.—The material was inoculated into tubes of litmus milk or Holman's meat medium, which was then heated to 80° C. for fifteen minutes, to eliminate the aerobic bacilli or nonspore-producing bacteria. They were then cultured, anaerobically, twenty-four, forty-eight, or seventy-two hours, or until growth appeared. Cultures were discarded if after four weeks growth did not appear. At the appearance of growth, subcultures were made on agar plates in an attempt to separate the mixed cultures. If distinct colonies were obtained on the agar plates, litmus milk, Holman's meat medium, and gelatin, an anaerobiosis was obtained by the following method:

The cultures in tubes or plates were placed in the evaporating jar with the lid ready to seal with Lubriseal. Within the jar, on a small hygroscopic earthenware dish, a flowerpot saucer, a piece of phosphorus, the size of a dime, was also placed. This was ignited and the lid sealed. This method gave the best results. Anaerobes, such as tetanus, which demand strict anaerobiosis, could be grown on the blood agar plate. The disadvantage of this method was the danger in handling the phosphorus, but by using special instruments and keeping it under xylol constantly, no difficulties were encountered. Rubber gloves were worn as an additional precaution.

Originally we used a Cenco-Hyvac pump attached to an improved Novy jar, but the vacuum obtained was so frequently lost over a twelve-hour period that this procedure was discarded in favor of the phosphorus method described. In this study particular attention was directed toward *Cl. welchii.* It was identified after pure cultures were obtained, using litmus milk, Holman's meat medium, and the blood agar plate. In case of doubt as to its identity, animal inoculation was carried out and cultures made from the animals. In some instances, attempts were made to demonstrate flagella, using Löffler's method. Other anaerobes were studied for motility. In most instances anaerobes in addition to *Cl. welchii* were found. Because of the difficulty at times of obtaining the anaerobe in question in pure culture, they were in the final analysis listed as unidentified anaerobic bacilli. In some instances a definite identification was possible following animal inoculation. The guinea pig was used because the rat was found to be too resistant to inoculation of any of the anaerobes. The mouse was also

resistant, but not to the same degree as the rat. The pigeon was not used. In some of the cases of spreading peritonitis a strain of *B. coli* was found that was particularly toxic for the mouse. The organism was cultured in bouillon, and the bouillon was filtered through a Berkefeld filter. Cultures were made from the filtrate to be sure of sterility. This strain produced a toxin which was fatal to mice in minute dilution. The toxin produced by certain strains of *Cl. welchii* was also studied. These toxins were prepared similarly to the method employed for obtaining the *B. coli* toxin. They were fatal to guinea pigs in small dilution, while in animals where the toxin was neutralized by perfringens antitoxin in similar dilution, the guinea pigs showed no harmful effects.

As a further proof of the pathogenicity of the strains of *Cl. welchii* which we encountered and as definite evidence of their etiologic activity in the toxemia of spreading peritonitis, samples of blood were collected from 35 patients who were convalescing or who had recovered from attacks of acute suppurative or gangrenous appendicitis or spreading peritonitis. In 73 per cent of the cases of peritonitis antitoxin was demonstrable in the patients' blood. In 10 normal adults, used as controls, no antitoxin was detected. This phase of our investigations is being reported in detail in another article.

In addition to the cultures made from the material removed from the peritoneal cavity of living dogs during the various stages of spreading peritonitis and at post mortem, cultures also were obtained at the initial operation from the lumen of the normal dog's appendix. Using a 5 c.c. Luer syringe and a small hypodermic needle, normal saline was injected into the lumen and withdrawn. This was cultured aerobically and anaerobically, using the technique previously described. The results obtained were then compared with the cultures obtained from the peritoneal cavity of dogs that had not been dead more than four hours. With one or two exceptions, the organisms obtained by culture, both aerobically and anaerobically, from the lumen of the normal appendix, were identical to those found in the peritoneal cavity at operation and at post mortem. Cultures of the heart blood in these cases were negative.

THE BACTERIOLOGY OF SPREADING PERITONITIS IN MAN

Specimens for cultures were removed from 55 patients suffering from gangrenous appendicitis associated with spreading peritonitis. Forty-one were positive for aerobes and 35 for anaerobes, a number of cultures showing both.

Of the aerobes, *B. coli* was present in 27, or 65.85 per cent; streptococcus in 16, or 39.02 per cent; other organisms were found only

occasionally and are listed in Table I. Multiplicity of associations was found in 72.97 per cent. *B. coli* was found alone more frequently than any other—10.53 per cent. The greatest number of different kinds of organisms in one culture was 6.

TABLE I

BACTERIAL FLORA OF SPREADING PERITONITIS IN MAN

AEROBES	PER CENT	ANAEROBES	PER CENT
B. coli	65.85	*Cl. welchii*	60
Str. non-haemolyticus	14.63	*Cl. welchii* alone	34.29
Str. viridans	14.63	*Cl. welchii* and other anaerobes	20
Str. faecalis	7.22	*Cl. welchii* and anaerobes and aerobes	40
Str. haemolyticus	2.44		
Gram-positive bacillus	29.27	Obligate anaerobes	5.71
B. subtilis	9.76	Gram-positive anaerobe unidentified	17.14
P. vulgaris	7.32	*B. putrificus*	8.57
Staph. aerogenes	4.88	*B. sporogenes*	5.71
B. proteus	2.44	Gram-positive diplococci	2.88
Gram-positive diplococci	4.88		
Gram-positive cocci	2.44		
Smegma bacillus	2.44		
Diphtheroid bacillus	2.44		
Staph. aureus	2.44		

From the 35 cultures in which anaerobes were obtained, *Cl. welchii* was present in 21, or 60 per cent; alone in 12, or 34.29 per cent; associated with other anaerobes in 7, or 20 per cent; with anaerobes or aerobes in 14, or 30 per cent. Other anaerobes were found only in scattering cases and are listed below. Multiplicity of association was present in 76.18 per cent. *Cl. welchii* was found alone most frequently, 23.81 per cent; with other anaerobes only in 9.52 per cent, and with aerobes in 66.61 per cent.

Cl. welchii was most frequently associated with *B. coli*, 52.38 per cent; with a Gram-positive aerobe, 23.81 per cent; with *Staph. albus*, 14.28 per cent; and with varieties of streptococci in 14.28 per cent.

THE BACTERIOLOGY OF INDUCED SPREADING PERITONITIS IN DOGS

Cultures were obtained from the peritoneal cavity of 154 dogs. These showed aerobic microorganisms in 130 instances; streptococci of various strains were present in 42, or 32.31 per cent; *B. coli* was present in 35, or 26.92 per cent; a Gram-positive aerobic bacillus in 25, or 19.23 per cent; *Staph. albus* in 8, or 6.15 per cent.

Anaerobes were associated with the aerobes in 89, or 68.46 per cent, of the 130 cultures. *Cl. welchii* was present in 82 of the 89, or 92.13 per cent; anaerobic streptococci in 6, or 6.74 per cent; and *B. sporogenes* in 1, or 1.12 per cent. *Cl. welchii* was associated with aerobes in 63.08 per cent of the 82 cultures. In 22, or 26.82 per cent, it was the only anaerobe found; in 60 instances, or 72.17 per cent, it was associated

with anaerobes and aerobes. In 5 instances anaerobes other than *Cl. welchii* were found alone; *B. putrificus* in 2; *V. septique* in 1; *B. histolyticus* in 1; and obligate anaerobe in 1.

Of the aerobes, *Cl. welchii* was most frequently associated with *B. coli*, in 35, or 42.68 per cent, of the 82 cultures; and with streptococci in 34, or 41.46 per cent.

Of the remaining anaerobes, a Gram-positive bacillus and an obligate anaerobe were found with equal frequency. The Gram-positive bacillus morphologically resembled *Cl. welchii*, but culturally it was not convincingly identified. In two of three instances anaerobic streptococci were associated with it. The obligate anaerobe on one occasion was associated with *B. putrificus*.

A COMPARATIVE STUDY OF THE BACTERIAL FLORA OF SPREADING PERITONITIS IN LIVING AND DEAD ANIMALS

The cultures obtained from man were, without exception, from the peritoneal cavity of the living. In order that a comparative study of the bacterial flora of peritonitis in the living human being might be made with those in the living dog, cultures were obtained from the peritoneal cavity of 18 dogs at varying periods following ligation of the appendix and the administration of castor oil. The results of these studies are shown in Tables II and III. Special interest in the

TABLE II

AEROBES	LIVING GROUP (PER CENT)	DEAD GROUP (PER CENT)
B. coli	14.28	28.92
Str. viridans	7.14	10.77
Str. non-haemolyticus	10.71	15.38
Str. hemolyticus	3.57	4.62
Str. faecalis		1.54
Gram-positive bacillus	28.58	19.23
Staph. albus	14.29	6.15
B. subtilis	14.29	6.15
B. proteus		5.39
Diplococci	3.57	3.85
Diphtheroids	3.57	

anaerobic aspect of the problem accounts in part for the fewer aerobic cultures. The percentage of component members of the aerobic living and dead groups is essentially the same. In the anaerobic living group obligate anaerobes, *B. sporogenes* and *B. histolyticus*, were not identified; while in the cultures taken from those who died, these microorganisms were noted on 15 different occasions. In the anaerobic living groups, however, Gram-positive anaerobes, not *Cl. welchii*, were encountered 11 times as compared with 3 times for the dead group. The greater incidence of *Cl. welchii* in the living group may account for the absence of obligate anaerobes in this group.

TABLE III

ANAEROBES	LIVING GROUP (PER CENT)	DEAD GROUP (PER CENT)
Cl. welchii alone	38.71	24.72
Cl. welchii and other anaerobes	29.03	44.94
Cl. welchii anaerobes and aerobes	29.03	22.47
Obligate anaerobes		13.48
Anaerobic streptococci	6.45	3.08
Gram-positive anaerobe, unidentified, but not *Cl. welchii*	6.45	2.31
B. putrificus	3.22	2.31
Sporogenes		0.77
V. septique	3.22	1.54
B. histolyticus		1.54
Gram-positive diplococcus	3.22	
Gram-positive diplococcic bacilli	3.22	
Gram-positive cocci	6.45	

A COMPARISON OF THE BACTERIAL FLORA OF SPREADING PERITONITIS IN MAN (LIVING) AND INDUCED SPREADING PERITONITIS IN DOGS (LIVING AND DEAD)

The incidence of *B. coli* is 144.61 per cent; Gram-positive aerobic bacilli, 52.21 per cent; and the streptococcus group, 20.85 per cent greater, respectively, in man than in the dog. The difference in percentage in the other groups is slight.

In the anaerobic group the incidence of *Cl. welchii* is 53.56 per cent greater in the dog. It was found alone more frequently in man than in the dog. The association with anaerobes is 4.46 per cent greater in the dog. *Cl. welchii* association with anaerobes and aerobes in the dog is only 6.51 per cent greater than in man. Gram-positive anaerobic bacilli

TABLE IV

PERCENTAGE OF MICROORGANISMS FOUND IN SPREADING PERITONITIS IN MAN (LIVING) AND IN INDUCED SPREADING PERITONITIS IN DOGS (LIVING AND DEAD)

AEROBES	MAN LIVING (PER CENT)	DOG LIVING AND DEAD (PER CENT)
B. coli	65.85	26.92
Str. non-haemolyticus	14.63	15.38
Str. viridans	14.63	10.77
Str. faecalis	7.32	1.53
Str. hemolyticus	2.44	4.61
Gram-positive bacillus	29.27	19.23
Staph. albus		6.15
B. subtilis	9.76	6.15
P. vulgaris	7.32	
Staph. aerogenes	4.88	
Gram-negative bacillus	4.88	
B. proteus	2.44	5.38
Gram-positive diplococci	4.88	3.85
Gram-positive coccus	2.44	
Smegma bacillus	2.44	
Diphtheroid bacillus	2.44	
Staph. aureus	2.44	

were found 205 per cent more frequently in the human being. In contrast to this, the obligate anaerobes in the dog were increased 175.48 per cent over those found in the human being. Anaerobic streptococci were not identified in man but were found in 8.74 per cent of instances in the bacterial flora of the dog.

TABLE V

ANAEROBES	MAN LIVING (PER CENT)	DOG LIVING AND DEAD (PER CENT)
Cl. welchii	60	63.07
Cl. welchii alone	34.29	28.83
Cl. welchii and other anaerobes	20	
Cl. welchii anaerobes and aerobes	40	73.17
Gram-positive anaerobe, unidentified	17.14	5.62
B. putrificus	8.57	3.37
B. sporogenes	5.71	1.12
Obligate anaerobe	5.71	15.73
Gram-positive diplococcus	2.86	
Anaerobic streptococcus		6.74

SUMMARY

The number and kinds of aerobes in spreading peritonitis in man are greater than those found in the dog. The greatest increase is in *B. coli*, 396.13 per cent. Four of the five members of the streptococcus group were found in the dog. This group was 81.7 per cent greater in the human being than in the dog. On the other hand, *Staph. albus* was found in the dog and *not* in man.

The most striking factor of the study is the close resemblance of the anaerobic flora in the two groups. In man *Cl. welchii* was found in 63 per cent. The only appreciable difference in percentage between the two groups, as concerns *Cl. welchii*, was in the association of this organism with other anaerobes and aerobes; in man this was 40 per cent as compared with 29.03 per cent in the dog. A Gram-positive anaerobe, not identified but not *Cl. welchii*, was found in 17.14 per cent in man and 6.45 per cent in the living dog. Anaerobic streptococci and *V. septique* were the only two important anaerobes identified in the cultures removed from the dog that were *not found in man.* Obligate anaerobes and *B. sporogenes* were found in the human cultures but *not in the dog.* The *B. histolyticus* was not found in either group. Gram-positive diplococci and cocci were not found in man.

CONCLUSIONS

The bacterial flora of spreading peritonitis in the living man and those of induced spreading peritonitis in the living and dead dog have been presented, together with a description of the methods used in obtaining the material and in studying the organisms.

The similarity between the microorganisms found in the lumen of the normal appendix and those recovered in spreading peritonitis is noted.

The findings of the comparative study of the various aerobic groups show a predominance of the *B. coli* in man as compared with the dog, using cultures made from the peritoneal cavity of both living and dead dogs.

A similarity of the anaerobic flora in man and in the dog, obtained from cultures from both living and dead animals, authenticates these findings. Of special significance is the presence of *Cl. welchii*, which apparently is present in the same ratio in spreading peritonitis *in human beings* as in induced spreading peritonitis *in dogs*.

REFERENCES

1. Laruelle, L.: Cellule **5**: 61, 1889.
2. Fraenkel, E.: München. med. Wchnschr. **37**: 23, 1890.
3. Jalaguier, M.: Bull. et mém. Soc. nat. de chir. **18**: 390, 1892.
4. Tavel, E., and Lanz, O.: Mitt. Arch. d. Klin. med. Inst. d. Schweiz. **1**: 1893.
5. Barbacci, O.: Centralbl. f. allg. Path. u. path. Anat. **4**: 968, 1893.
6. Welch, W. H., and Flexner, S.: J. Exper. Med. **1**: 5, 1896.
7. Tavel, E., and Lanz, O.: Rev. de chir. **30**: 43, 215, 1904.
8. Veillon and Zuber: Arch. de Med. Exper. **10**: 517, 1898.
9. Welch, W. H.: Bull. Johns Hopkins Hosp. **40**: 186, 1900.
10. Krogius: Jena, 1901.
11. Lanz, O., and Tavel, E.: Rev. de. chir. **30**: 43 and 215, 1904.
12. Hewetson, John T.: Brit. M. J. **2**: 1457, 1904.
13. Dudgeon, Leonard S., and Sargent, P. W. G.: The Bacteriology of Peritonitis, London, p. 100, 1905, Archibald Constable & Co.
14. Manahan, Thomas J.: Boston M. & S. J. **152**: 346, 1905.
15. Heyde, M.: Beitr. z. klin. Chir. **76**: 1, 1911.
16. Runeberg, B.: Brun's Beitr. z. klin. Chir. **78**: 130, 1911.
17. Vick, M.: Saint Bartholomew Hospital Reports **48**: 97, 1912.
18. Fishbein, Morris: Am. J. M. Sc. **144**: 502, 1912.
19. Deaver, John B.: Appendicitis; Its Diagnosis and Treatment, Philadelphia, 1913, P. Blakiston's Son & Co.
20. Meleney, F. L., Harvey, H. D., and Zaytseff-Jern, Helen: Arch. Surg. **22**: 1, 1931.
21. Kelly, H. A., and Noble, C. P.: Gynecology and Abdominal Surgery, Philadelphia, 1914, W. B. Saunders Co.
22. Ashurst, A. P. C.: Surgery, Principles and Practice, Philadelphia, 1920, Lea and Febiger, p. 854.
23. Murphy, John B.: Appendicitis, Keen's Surgery, Philadelphia, 1921, W. B. Saunders Co., p. 4.
24. Sherrill, J. Garland: Peritonitis, New York, 1925, D. Appleton-Century Co.
25. Royster, H. A.: Appendicitis, New York, 1927, D. Appleton-Century Co.
26. Weinberg, M., Pierot, A. R., Davesne, J., and Renard, Claud: Recherches sur la bacteriologie et la serotherapie des appendicitis à aigue, Ann. Inst. Pasteur **42**: 187, 1928.
27. Meleney, F. L., Olpp, John, Harvey, H. D., and Zaytseff-Jern, Helen: Arch. Surg. **25**: 703, 1932.
28. Homans, John: Practice of Surgery, Springfield, 1933, Charles Thomas.
29. DaCosta, J. Chalmers: Modern Surgery, Philadelphia, 1931, W. B. Saunders Co.
30. Romanis, W. H. C., and Michiner, P. H.: Science and Practice of Surgery, Baltimore, 1932, William Wood & Co.
31. Boyd, Wm.: Surgical Pathology, Philadelphia, 1933, W. B. Saunders Co.
32. Nelson's Loose-Leaf Surgery, New York, 1933, Thomas Nelson & Sons.

CALCIFIED FECALITH IN THE APPENDIX

Report of a Case of Acute Suppurative Appendicitis Perforated by a Calcified Fecalith

Harold J. Shelley, M.D., New York, N. Y.

(From the Surgical Service and Gastro-Intestinal Clinic, St. Luke's Hospital)

THIS case is reported not only because calcified fecaliths in the appendix are quite unusual but also because undoubtedly this fecalith was the cause of the perforation of the appendix. Had a diagnosis of the condition been made from a roentgenogram made more than four months before the onset of the attack of acute suppurative appendicitis, the patient would have been spared the ordeal through which he went and which almost cost him his life.

CASE REPORT

In December, 1936, the patient had a small papilloma of the urinary bladder which was successfully removed by fulguration. At that time a plane plate of his abdomen was taken (Fig. 1). The triangular shadow in the right lower quadrant was overlooked at that time and was not noted until subsequent review after the fecalith had been discovered at the time of operation four months later.

On April 18, 1937, he was admitted to the medical ward at St. Luke's Hospital because of generalized abdominal pain of one week's duration. This had started as a dull constant pain in the epigastrium which had no relation to food-taking and did not radiate. Laxatives and enemas had given temporary relief. At the end of six days, the pain shifted to the lower abdomen and became so severe that the patient was frequently doubled up with pain. It was definitely of a colicky nature. He became nauseated and vomited. There was a fever of unknown degree for the three days before admission. Enemas had given good results until two days before admission, after which time they were almost entirely unsuccessful. In these latter two days he had developed a moderate degree of abdominal distention.

Physical examination after admission showed no rigidity of the abdominal muscles. Slight tenderness was present in the left lower quadrant and a moderate degree of distention presented. No tenderness was found by rectal examination. The rectal temperature was 101° F. and pulse was 100. The leucocyte count was 8,000 per c. mm., with a differential of polymorphonuclears, 72; lymphocytes, 24; and large mononuclears, 4.

The day after admission his rectal temperature was 101.4°; pulse, 110; and respirations, 24. The leucocyte count had increased to 12,300 per c. mm. with a differential of polymorphonuclears, 92; lymphocytes, 8; and a moderate shift to the left. The abdomen was now quite markedly distended with generalized rigidity and tenderness, neither of which presented any localization.

A roentgenogram showed marked distention of loops of small intestine (Fig. 2). Again the shadow of the fecalith was present, but it was not recognized until the film was re-examined after the operation.

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Fig. 1.

Fig. 2.

Fig. 1.—Roentgenogram made in December, 1936. The calcified fecalith can be seen lying in the right lower quadrant. At the time this film was made, there was nothing in the history or physical examination to point to trouble in the appendix.

Fig. 2.—Roentgenogram made on April 19, 1937. The calcified fecalith still shows in the right lower quadrant although in a slightly different position from that in the preceding film. At this time the film also revealed the presence of several distended loops of small intestine.

Fig. 3.—The appendix as it appeared after removal. The fecalith shows as it presented at the time of operation. The slit in the wall toward the mesoappendix was made in removing the fecalith from the appendix for examination.

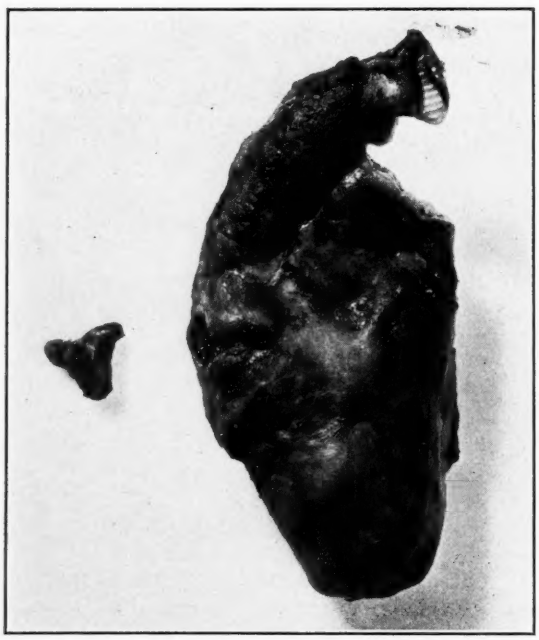

Fig. 4.—The appendix with the fecalith as it appeared after removal from the appendix. The original round perforation can be seen at the extreme left. The slit toward the mesoappendix was made in removing the fecalith.

A surgical consultation was requested. Immediate operation was advised with a preoperative diagnosis of generalized peritonitis, probably appendical in origin.

Under gas oxygen ether anesthesia a right lower median rectus incision was made. A McBurney incision was not made as there had not been sufficient localizing signs or symptoms to point to the location of the appendix or other possible site of the origin of the peritonitis. Upon opening the peritoneum, a considerable amount of foul-smelling purulent fluid, containing gross particles of fecal material, was found. A large gangrenous appendix, about which were wrapped coils of small intestine, lay just behind and to the right of the umbilicus. Near its middle, opposite the mesenteric attachment, was a perforation from which projected the point of a fecalith (Fig. 3).

The mesoappendix was clamped, cut, and ligated. The base of the appendix was divided between clamps and the stump inverted with two 00 chromic catgut purse-string sutures without ligating. Two rubber dam drains were inserted into the peritoneal cavity, one into the pelvis and one into the right gutter. The wound was closed in layers with interrupted chromic sutures and three silkworm tension sutures which included the skin, subcutaneous tissues, and rectus sheath. The skin was closed with interrupted 00 chromic catgut.

Pathologic Examination.—The specimen consists of an appendix 8 cm. long and 3 cm. in diameter. The surface is hemorrhagic and covered with a fibrinopurulent exudate. In the midportion is a perforation through which a fecalith is protruding. On cut section the wall is greatly thickened and all of the walls are involved in a marked inflammatory reaction. The lumen contains purulent material.

Microscopic examination shows the appendix to have undergone extensive suppuration with perforation of the wall and destruction of most of the mucosa, but also with a widespread productive lesion which has walled off most of the suppuration. The surface is thickly coated with a large amount of cellular granulation tissue but there remains a large amount of purulent exudate in the appendix.

Diagnosis.—Acute suppurative apendicitis with perforation. Calcified fecalith.

Postoperative Course.—Convalescence was very stormy for the first ten days. The first week the temperature ranged from 100 to 103.4°, with the pulse ranging from 100 to 116. The drains were gradually shortened and were removed on the sixth day. For the first nine days there was a complete mechanical obstruction. During this time the distention was well controlled by the continuous use of a Levin tube. The patient was carried along on glucose and normal saline solution which was given intravenously.

Through the second week the temperature ranged between 99.4 and 101°, with a pulse under 96. The wound suppurated and broke down completely to the peritoneum which remained closed. This was cared for by adhesive strapping. After the second week, the bowels moved well and the patient was able to take a normal amount of food and fluids. The pulse and temperature became normal. The wound healed firmly, after cleaning up nicely.

When discharged on June 8, 1937, he was up and about, entirely symptom-free. Up to the time when last seen, eleven months after the operation, he had had no complaints or difficulties of any kind. He wears an abdominal belt. The scar is firm and, since six months after the operation, he has played tennis, being careful not to exert himself too strenuously.

SUMMARY AND DISCUSSION

A case is reported in which a calcified fecalith was found in the appendix. This might have been recognized in a roentgenogram made four months before it resulted in a very serious illness. Removal of

the appendix would have spared the patient this illness which easily might have resulted in his death. An attack of acute suppurative appendicitis subsequently developed with perforation of the appendix by the fecalith. Up until a typical picture of generalized peritonitis presented itself, the patient had appeared to have only an obscure and indefinite intraabdominal condition.

Appendectomy with drainage was performed. A mechanical intestinal obstruction, which was present from the day of the operation, was controlled for nine days by the use of a Levin tube. The obstruction then opened spontaneously. Notwithstanding the fact that the wound broke down completely to the peritoneum, a firm scar was obtained by the use of adhesive strapping. The patient has had no constipation or indigestion since the operation.

The fecalith showed in two roentgenograms of the abdomen, one made four months before and the other the day before the operation. In 1916 Douglas and Le Wald[1] reported in the *Journal of the American Medical Association* 3 cases in which calcified fecaliths had been demonstrated by roentgen examination. Previously, Weisflog,[2] in 1906, had reported 1 case; Seelig,[3] 1 case in 1908; Pfahler and Stamm,[4] 2 cases in 1915; and Hunner,[5] 1 case; Stewart, [6] 1 case; Case,[7] 2 cases; Eastmond,[8] 1 case, all in 1916. The cases of Case and Eastmond were all three diagnosed ureteral calculi before operation but were found to be calcified fecaliths in the appendix.

REFERENCES

1. Douglas, John, and Le Wald, Leon T.: Fecal Concretions of the Appendix, J. A. M. A. **66**: 1919-1920, 1916.
2. Weisflog: Zur roentgenographischen Diagnose der Enterolithen des process. Vermiformis, Fortschr. a. d. Geb. d. Röntgenstrahlen, Hamb. **10**: 217-219, 1906.
3. Seelig, M. G.: Appendicitis Resembling Ureteral Calculus, Surg., Gynec. & Obst. **7**: 485-486, 1908.
4. Pfahler, G. E., and Stamm, C. J.: Diagnosis of Enteroliths by Means of the Roentgen Ray, Surg., Gynec. & Obst. **21**: 14-17, 1916.
5. Hunner, G. L.: Stricture of the Ureter, New York M. J. **104**: 5-11, 1916. (Discussion by Le Wald: New York M. J. **104**: 817, 1916.)
6. Stewart, W. H.: Quoted from Douglas and Le Wald.[1]
7. Case, J. T.: Quoted from Douglas and Le Wald.[1]
8. Eastmond, Charles: Quoted from Douglas and Le Wald.[1]

THE SURGICAL TREATMENT OF COMPLICATED DUODENAL ULCER

WILLIAM L. WOLFSON, M.D., AND ROBERT E. ROTHENBERG, M.D.,
BROOKLYN, N. Y.

THE surgical treatment of duodenal ulcer is a contentious subject which has long received contemplative thought, thorough discussion and much surgical application.

Various and ingenious operations have been tried to obtain a satisfactory and lasting cure for peptic ulcer. Excision of the ulcer, pyloroplasty, gastroduodenostomy, gastrojejunostomy, the von Eiselsberg or Devine exclusion, partial gastrectomy, or combinations of these operations are the measures most frequently used. Each of these procedures for duodenal ulcer has proponents who give substantial reasons and highly favorable advantages for their choice. Because these preferred operations are often so anatomically different and the disputation over their respective value and results so vigorous, it may be sensed that the surgical solution of the ulcer problem is not completely determined.

It may be generally stated that to cure peptic ulcer the gastric hyperacidity and the excess pepsin secretion must be controlled, and most of the controversy involves the question of which surgical measure is best suited to accomplish this result. The conservative point of view is expressed in the remark of C. H. Mayo:[1] "If any man wanted to cut out half of my good stomach in order to cure a little ulcer of my duodenum, I would run faster than he"; and also that of Lord Moynihan,[1] who stated: "Gastrectomy for duodenal ulcer is neither safe nor simple, nor does it bring an adequate reward." On the other hand, the more radically minded surgeons agree with Lahey's[2] statements: "It is certain in my mind that jejunal ulcer is a too frequent, too difficult and too dangerous a post operative sequela to gastroenterostomy to permit the operation to be employed as a routine surgical method of treating particularly duodenal ulcer. I think all of us owe to Finsterer, Haberer, Strauss, Berg and others a debt of gratitude for adhering in the face of criticism to the position that subtotal gastrectomy is the best operation for patients with intractable peptic ulcer."

At this time it may well be asked which of these two diametrically opposed positions is the more correct. Most surgeons are inclined to agree that each case of duodenal ulcer should receive individual consideration and planning and that no one surgical procedure is appli-

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cable to all cases. There is now, however, sufficient clinical evidence that intractable ulcers, either in the stomach or duodenum, are difficult to cure if the gastric mucosa continues to secrete or if it maintains an overabundance of hydrochloric acid and pepsin. The wider acceptance of this premise has encouraged the more frequent application of subtotal gastrectomy to those patients who have failed to respond to thorough medical management.

The popularity and utility of gastroenterostomy for duodenal ulcer are mainly based on fair results, ease of performance, and low operative risk. Unfortunately, however, exceedingly difficult secondary operations are too often required after gastroenterostomy failures because of symptoms from the original ulcer or newly formed ulcers, or particularly from the frequent formation of a gastrojejunal ulcer. The most radical operation used to overcome and cure an intractable ulcer in the first or second portion of the duodenum is a gastroduodenal resection beyond the ulcer; but, if this effort brings too high a mortality, then a gastroenterostomy with all its postoperative disappointments would seem the wiser course. It is important, therefore, that, when making a comparative study of results, a carefully considered estimate be made of the combined hazards of gastroenterostomy plus a secondary operation, against the single danger of one gastric resection!

The exclusion and division of the stomach with gastrojejunostomy initiated by von Eiselsberg and Devine, although it has certain advantages and appeal, is not simple to perform nor are the end results entirely satisfactory, for there is left behind a gastric mucosa which continues to secrete large amounts of acid. Moreover, it has been reported that the operation is followed by a high incidence of jejunal ulcers.

If the ulcer is in the first portion of the duodenum and has not become adherent to surrounding structures, it is good practice to resect that area of the duodenum with partial gastrectomy to a region distal to the ulcer. However, when the ulcer is located far down in the second part of the duodenum, when the lesion is adherent, deep seated, or penetrating into the pancreas, if it impinges upon the region of the common bile duct or is surrounded by a large inflammatory exudate, resection of these tissues is most dangerous. Pancreatitis, common bile duct injury, duodenal fistula or peritonitis are formidable complications which may follow an attempt to remove the first and part of the second portions of the duodenum in the presence of such pathology.

To avoid the possibility of these complications, the involved duodenum, pyloric sphincter, and pyloric area are occasionally not removed when subtotal gastric resection is done. Retention of an intact

pylorus, however, may defeat the purpose of a resection operation because the glands in the pyloric mucosa by hormonal action may stimulate the glands in the fundus to produce acid. Furthermore, the secretions of the mucosa, locked in by the pyloric sphincter below and the closed end of the pylorus above, may accumulate in sufficient quantity and may develop enough tension to burst the suture line. Another serious and technical objection to this conservative kind of prepyloric subtotal gastric resection is the difficulty frequently encountered in securely closing the pyloric stump because the gastritis which accompanies duodenal ulcer usually produces much edema and inflammatory reaction in the mucosal wall of the pyloric region.

The desideratum then would appear to be an operation which will not risk the extremely high mortality of a gastroduodenal resection beyond the ulcer into the region of the bile ducts or pancreas, but which will remove sufficient stomach to produce or approximate an achlorhydria. We would like to emphasize the fact that such penetrating adherent ulcers are better left in situ, for healing usually occurs if the gastric current is completely diverted and if an achlorhydria can be approached.

Finsterer, in 1918, devised and has continued to use an "in between" operation which he terms "resection for exclusion." The procedure is a compromise between subtotal gastrectomy and the Devine or von Eiselsberg exclusion operation. Without resecting the ulcer-bearing portion of the duodenum, it yields the same satisfactory results as the subtotal gastrectomy with less morbidity and mortality. Finsterer and Cunha[3] in describing "resection for exclusion" wrote: "In a case in which there is well marked hypertrophy of the musculature the line of separation of the antrum must be farther away from the pylorus in order to make the Lembert sutures without any stretching or tension. In these cases the mucous membrane from the eliminated portion of the antrum is removed as far as the pylorus and there closed by a suture. The seromuscularis is cut on the side of the greater and lesser curvature making thus an anterior and a posterior flap which is overlapped and fastened by suture over the mucous membrane suture in order to make a safe and sure closure." Bancroft[4] accomplishes the same result, but his procedure differs from the above in that he first divides the stomach near the reentrant angle and then begins his seromuscular dissection downward toward the pylorus. This entails much more dissection, carried out with the stomach opened, and more soiling and leaves a surplus of seromuscular wall to be inverted after the mucosa has been removed therefrom.

Our own experience is that subtotal gastrectomy or some modification thereof is the method of choice for ulcer cases with unusually high gastric acidity, in those who have had repeated hemorrhages, and in those who have an advancing ulcerative process. Also, the post-

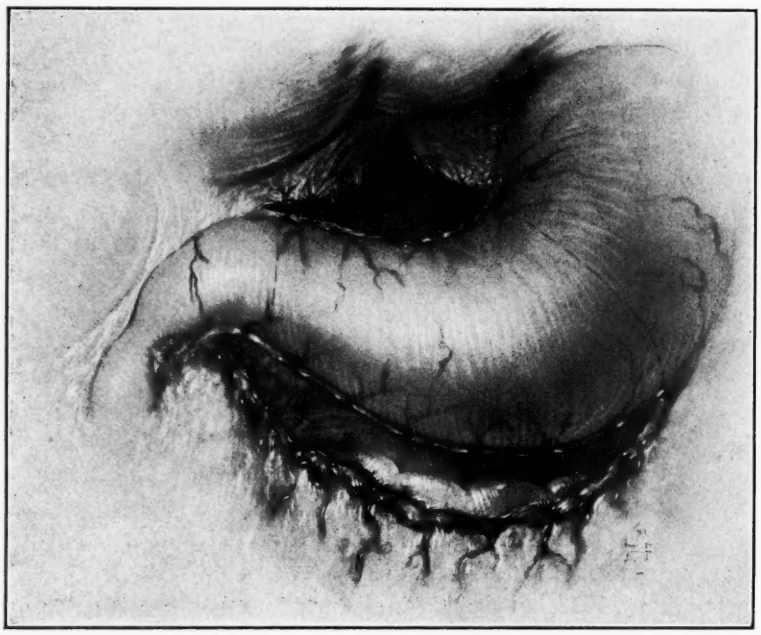

Fig. 1.—The stomach freed from gastrohepatic and gastrocolic ligaments. Lines indicate area of resection.

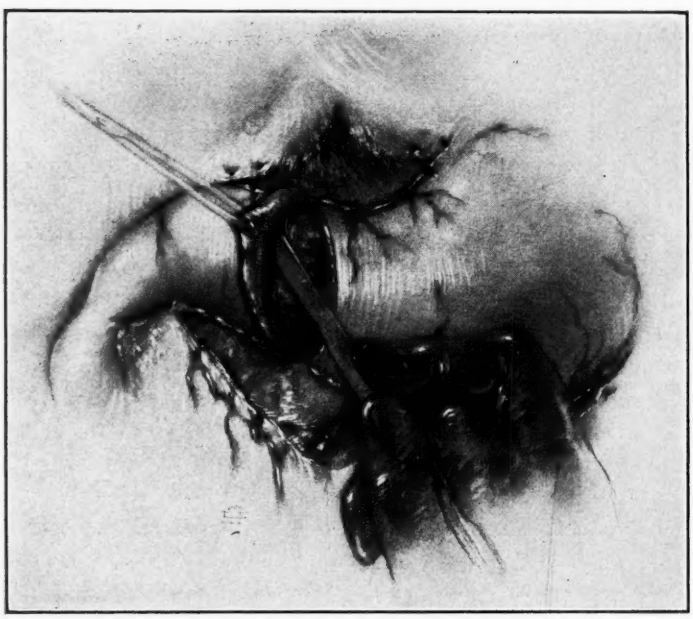

Fig. 2.—Seromuscular flap dissection starting 1½ inches above the pyloric vein.

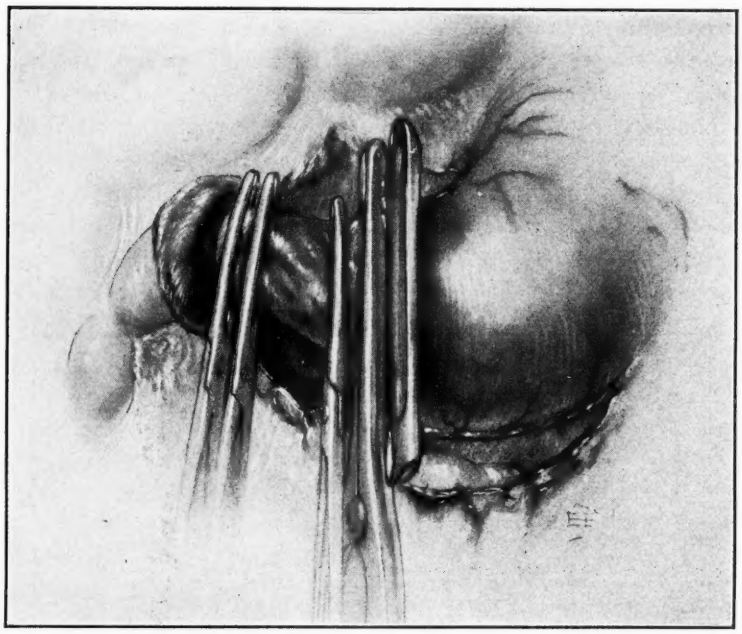

Fig. 3.—Excision of the unopened tube of pyloric mucosa. The proximal clamp is a Payr clamp with a hinged cover.

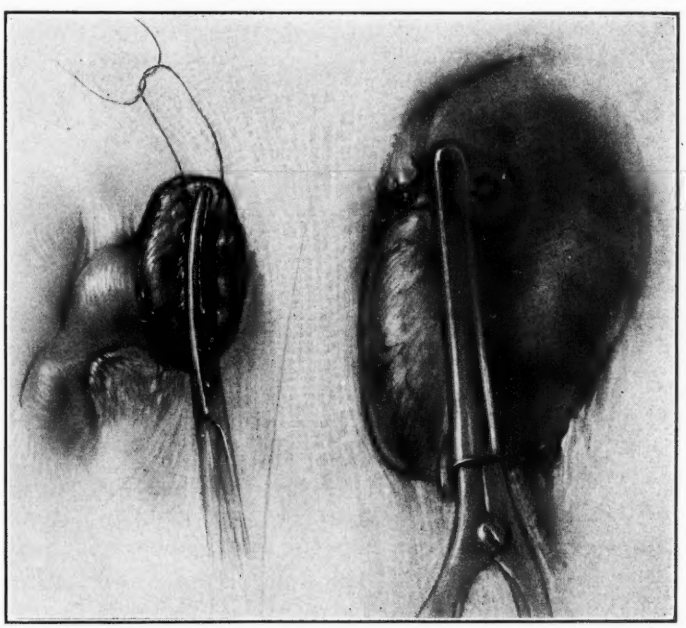

Fig. 4.—A purse-string suture infolds the mucous membrane at the pyloric sphincter. Payr clamp with shield protects the cut end of stomach.

operative persistence of symptoms, the possibility of recurrent hemorrhage, and the fear of marginal ulcer after gastroenterostomy are important factors which have influenced us toward partial stomach resection. For selected cases of penetrating adherent ulcers of the duodenum, we have been doing a conservative prepyloric subtotal gastrectomy, the description of which is as follows:

An incision is made to the right of the midline from the xiphoid process to about the level of the umbilicus. The peritoneal cavity is opened. The stomach is brought into view. The gastrocolic omentum is separated from the greater curvature of the stomach from the region

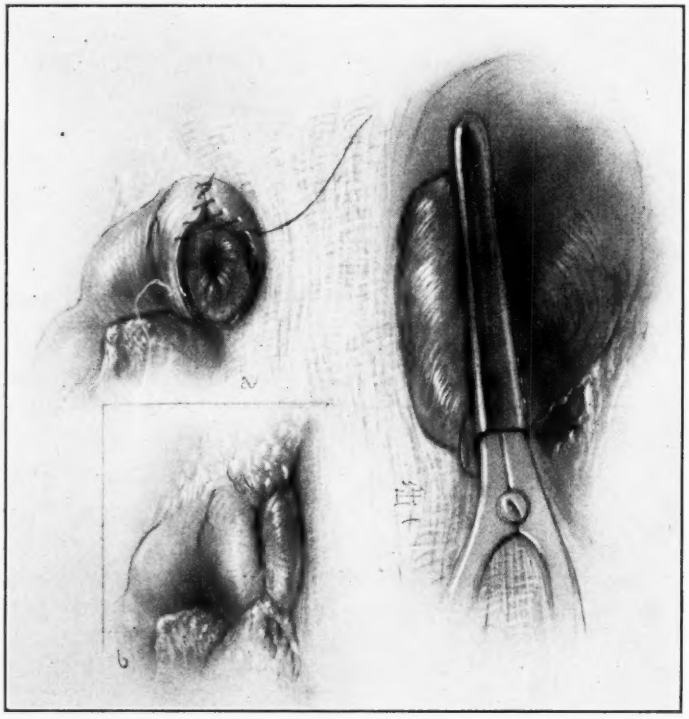

Fig. 5.—*a*, Closing the pyloric stump with a continuous suture; *b*, tabs of omentum used to reinforce the closure.

of the pyloric sphincter to beyond a point on the fundus at which resection will be carried out. The dissection is made as close to the stomach wall as possible in an endeavor to leave behind a maximum amount of the blood supply to the omentum. The hand is then passed behind the stomach and the gastrohepatic ligament is put on the stretch. The lesser curvature of the stomach is freed from its attachments by ligating and severing the vessels which traverse the gastrohepatic omentum. During these two steps of the operation, branches of the left gastroepiploic, the right gastroepiploic, and the right and left gastric vessels are ligated and cut (Fig. 1). An incision is then

made through the serous and muscular coats of the stomach approximately 1½ inches above the pyloric sphincter. This incision completely encircles the stomach at this level. By blunt dissection between the mucosal and muscular layers, a cuff of serosa and muscle is raised and is easily pushed back from the line of incision to the pyloric sphincter (Fig. 2). The dissection is stopped when it reaches the pyloric ring where the mucosa becomes intimately attached to the muscularis. The unopened tube of mucosa is doubly clamped at each end and cut across (Fig. 3). The proximally applied clamp is a hinged cover, Payr clamp. A purse-string suture is placed within the seromuscular flap immediately above the pyloric ring. This step completely buries the edge of the mucosa and prevents bleeding therefrom (Fig. 4). The cuff of seromuscularis of the pylorus is now easily infolded and securely closed by continuous suture (Fig. 5). The stomach is then resected at a level which will remove about three-quarters of the lesser curvature and at least two-thirds of the greater curvature (Fig. 1). A retrocolic isoperistaltic anastomosis is carried out between the cut end of the stomach and a high loop of jejunum. When completed, the operation leaves us with a closed stump of pylorus minus its mucosa, an excluded ulcer left in situ, and a high gastric resection with a jejunal anastomosis.

SUMMARY

1. The surgical treatment of complicated duodenal ulcer is outlined.

2. The dangers of radical gastroduodenal resection carried beyond an involved duodenal ulcer are emphasized.

3. A plea for subtotal gastrectomy of the Finsterer type is made.

4. A conservative modified subtotal prepyloric gastrectomy is briefly described. The details of handling the pyloric stump and its mucosa are given.

Payr clamp described is made by J. Sklar Mfg. Co., Brooklyn, N. Y.

REFERENCES

1. Mayo, C. H., and Lord Moynihan: Quoted by Rowlands and Turner, Operations of Surgery, Wm. Wood & Co., Baltimore, 1937, Vol. II, p. 154.
2. Lahey, F. H.: S. Clin. North America **15**: 1401-1413, 1935; Am. J. Dig. Dis. & Nutrition **2**: 673, 677, 1935-36.
3. Finsterer, H., and Cunha, F.: Surg., Gynec. & Obst. **52**: 1099-1114, 1931.
4. Bancroft, G. L.: Am. J. Surg. **16**: 223-230, 1932.

PRIMARY CARCINOMA OF THE BILIARY SYSTEM

A Clinicopathological Analysis of Forty Cases

Rigney D'Aunoy, M.D., Michael Alexander Ogden, M.D., and Béla Halpert, M.D., New Orleans, La.

(From the Departments of Pathology and Bacteriology, Charity Hospital of Louisiana at New Orleans, and the School of Medicine, Louisiana State University Medical Center)

THE present study is based on the clinical records, the necropsy findings, and the gross and microscopic appearances of preserved specimens in 40 cases of primary carcinoma of the biliary system encountered among 6,050 necropsies on persons over one year of age performed by the staff of the Department of Pathology of the Charity Hospital of Louisiana at New Orleans, between January 1, 1931, and October 6, 1937. It is particularly concerned with the site of the primary growth, its structure, its spread locally and to distant parts, its clinical manifestations and their duration, and the immediate cause of death in the afflicted patients.

SITES OF PRIMARY GROWTHS; DISTRIBUTION ACCORDING TO RACE, SEX, AND AGE

Of the 40 tumors, 23 were primary in the liver; 6, in the extrahepatic biliary ducts; and 11, in the gallbladder. Twenty of the patients were negro (18 male, 2 female) and 20 were white (12 male and 8 female). One of the patients died in the second, 3 in the fourth, 8 in the fifth, 11 in the sixth, 14 in the seventh and 3 in the eighth decade of life. The youngest patient was thirteen years of age and the oldest was 77 years of age (Table I).

STRUCTURE OF NEOPLASMS

Of the 23 primary liver neoplasms, 16 were liver cell and 7 were cylindrical cell carcinomas. All of those primary in the extrahepatic biliary ducts and gallbladder were cylindrical cell carcinomas.

In 7 cases of liver cell carcinomas a tumor mass was located in the right lobe of the organ. In 2 of these the mass was single, measuring 2.5 cm. in diameter in one instance and 8 cm. in the other. In the 5 others single tumors measuring up to 20 cm. in diameter with numerous smaller ones throughout the organ were present. In the remaining 9 cases of liver cell carcinomas, various sized tumor nodules up to 4 cm. in diameter were scattered throughout the liver, which was usually enlarged, generally weighing over 3,000 gm. A varying degree of fibrosis

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was present in all of these livers. Central areas of necrosis and hemorrhage were noted in the medium-sized and large nodules, and umbilication in those near the surface.

In 1 case of the 7 cylindrical cell carcinomas primary within the liver, there were 2 tumor nodules, 2.5 and 5 cm. in diameter. In the other 6 cases there was fairly even distribution of small tumor nodules, few over 2 cm. in diameter, in enormously enlarged livers weighing up to 7,000 gm.

In the 6 cases of carcinoma of the extrahepatic biliary ducts, the primary growths were small, not over 1 cm. in diameter. In 4 of these the common bile ducts and in 2 the common hepatic ducts were the site of the growth. In all 6 cases, however, the neoplasms obstructed the duct lumens, causing dilatation of their proximal portions with resultant jaundice. All but 1 of these cases had calculi in the gallbladders.

In the 11 cases of primary carcinoma of the gallbladder, the walls of the organ were thickened by neoplastic infiltration. A fungating mass protruded into the cavity of the organ in 4 of the cases, and biliary calculi (mixed gallstones) were present in 7.

The microscopic structure of the liver cell carcinomas varied. Usually sheets of polygonal cells resembling huge liver cells, with a stroma of delicate-walled capillaries, formed the main bulk of the growth. The body of these cells stained deep pink with occasional light stippling (hematoxylin and eosin). One, frequently two, and occasionally more, large, vesicular, deeply staining nuclei occupied their centers. A number of the nuclei were in mitosis. In some areas, particularly near capillaries or at the periphery of the growth, the cell borders were distinct, the cells often being arranged in cords of various lengths or masses enclosing slitlike lumina, these containing a golden yellow, granular material suggestive of bile. This pattern was usually mimicked by the metastases occurring in the regional lymph nodes and remote organs (Fig. 1). Usually towards the center of the growth the cell borders of the tumor cells were faint, and large pale pink staining cytoplasmic masses occupied the spaces between well-preserved, clearly outlined, tumor cells. Areas of necrosis and hemorrhage were frequent, especially in the larger tumor masses. In some instances a delicate, loose connective tissue stroma was conspicuous. Usually, however, broad, dense fibrous connective tissue septa subdivided or surrounded the growths. The stroma was generally infiltrated with numerous small round cells, some large mononuclear cells, and occasionally with polymorphonuclear leucocytes. Islands of liver cells regularly remained preserved amidst the sheets of tumor cells (Fig. 2) or appeared as compressed narrow cords. Sometimes the growth was well circumscribed and sharply demarcated from the surrounding tissue. Vessels containing tumor thrombi were frequently encountered. In other instances the growth was partly surrounded by a zone of dense hyalinizing

TABLE I

PATIENT #	AGE	SEX AND RACE	SPREAD		CLINICAL MANIFESTATIONS	CIRRHOSIS OF LIVER CHOLELITHIASIS	DURATION OF ILLNESS MONTHS	CAUSE OF DEATH
			REGIONAL	DISTANT				
1. Patients with Liver Cell Carcinomas								
L-1 '36—1005	13	M W	Lymph nodes; peritoneum	Lungs	Weight loss; jaundice; ascites	Cirrhosis	16	Carcinoma
L-2 '36—1296	35	M C	Lymph nodes	Lungs	Ascites	Cirrhosis	3	Thrombosis, vena cava inferior
L-3 '35— 16	47	M C	Lymph nodes	Lungs	Jaundice; ascites	Cirrhosis	2	Thrombosis, vena portae
L-4 '31— 286	48	M C	None	None	Weight loss; jaundice; ascites	Cirrhosis	5	Thrombosis, vena portae
L-5 '34— 63	49	M C	None	Lungs	Jaundice; ascites	Cirrhosis	4	Carcinoma
L-6 '33— 827	51	M C	None	None	Weight loss; jaundice; ascites	Cirrhosis		Hemorrhage, intraperitoneal
L-7 '32— 666	53	M W	None	None	Jaundice	Cirrhosis	3	Cirrhosis of liver
L-8 '33— 450	56	M C	Lymph nodes	Lungs	Jaundice; ascites	Cirrhosis	13	Thrombosis, vena cava inferior
L-9 '35— 68	56	M W	None	Lungs	Jaundice; ascites	Cirrhosis	2	Carcinoma
L-10 '37— 336	58	M W	Lymph nodes; peritoneum	Lungs; suprarenals	Weight loss; ascites	Cirrhosis	3	Carcinoma
L-11 '37— 166	61	M C	None	None		Cirrhosis		Nephritis, suppurative
L-12 '37— 732	64	M W	Lymph nodes; peritoneum		Weight loss; jaundice	Cirrhosis	6	Carcinoma
L-13 '31— 531	64	M W	None	None	Jaundice; ascites	Cirrhosis	2	Cirrhosis of liver
L-14 '37— 187	65	F W	None	Lungs	Jaundice; ascites	Cirrhosis	4	Pneumonia, diffuse
L-15 '31— 658	65	M C	None	None	Weight loss; jaundice; ascites	Cirrhosis; cholelithiasis	3	Carcinoma
L-16 '33— 701	74	M C	None	Lungs	Weight loss	Cirrhosis	4	Carcinoma
2. Patients with Cylindrical Cell Carcinomas Primary Within the Liver								
C-1 '37— 700	37	M C	None	None	Weight loss		4	Tuberculosis of lungs
C-2 '36— 719	42	M C	Lymph nodes	Lungs	Ascites	Cirrhosis; cholelithiasis		Carcinoma
C-3 '34— 729	46	F W	Lymph nodes	Lungs	Jaundice; ascites	Cirrhosis	3	Carcinoma
C-4 '33— 481	52	M C	None	Lungs	Jaundice; ascites	Cirrhosis	4	Carcinoma
C-5 '37— 736	54	F W	None	None	Weight loss; jaundice; ascites	Cirrhosis	2	Hemorrhage, gastric
C-6 '37— 941	64	M W	None	None	Weight loss	Cirrhosis	3	Carcinoma
C-7 '34— 777	67	F W	None	None	Jaundice; ascites	Cirrhosis		Cirrhosis of liver

TABLE I—CONT'D

3. Patients with Cylindrical Cell Carcinomas Primary in the Extrahepatic Biliary Ducts									
D-1	'32—1036	44	M	W	Liver	None	Jaundice; ascites	Cirrhosis; cholelithiasis	3	Carcinoma
D-2	'31— 730	49	F	C	Lymph nodes; gallbladder	None	Jaundice	Cholelithiasis		Pneumonia, focal
D-3	'33— 255	62	F	W	Liver; pancreas	None	Weight loss; jaundice	Cholelithiasis	2	Pneumonia, focal
D-4	'34— 32	66	M	C	Gallbladder	None	Weight loss; jaundice			Hemorrhage, gastric ulcer
D-5	'34— 849	68	M	W	Lymph nodes; liver; pancreas	Lungs	Weight loss; jaundice; ascites	Cholelithiasis	4	Carcinoma
D-6	'33— 656	76	F	W	Lymph nodes; liver	Lungs; kidneys	Weight loss; jaundice	Cholelithiasis	9	Carcinoma
4. Patients with Cylindrical Cell Carcinomas Primary in the Gallbladder									
G-1	'35— 194	32	M	W	Lymph nodes; liver	None	Jaundice; ascites	Cirrhosis; cholelithiasis	4	Pneumonia
G-2	'33—1218	43	M	C	Liver; peritoneum		Ascites	Cholelithiasis	2	Carcinoma
G-3	'35—1267	51	F	C	Liver	Lungs		Cirrhosis; cholelithiasis	4	Carcinoma
G-4	'33—1266	51	M	C	Lymph nodes; liver	Spleen	Jaundice; ascites	Cirrhosis; cholelithiasis	2	Carcinoma
G-5	'31— 665	56	F	W	Lymph nodes	None	Jaundice; ascites	Cholelithiasis	12	Peritonitis, generalized
G-6	'31— 58	60	M	C	Liver	None	Jaundice	Cirrhosis	3	Carcinoma
G-7	'33— 371	65	M	C	Lymph nodes	None	Weight loss	Cirrhosis	6	Peritonitis, generalized
G-8	'36— 186	67	F	W	Liver	None	Weight loss		2	Nephritis, suppurative
G-9	'31— 256	70	M	W	None	None	Jaundice; ascites	Cirrhosis; cholelithiasis		Cirrhosis of liver
G-10	'37— 175	70	M	W	None	None	Jaundice	Cirrhosis; cholelithiasis	4	Carcinoma
G-11	'33— 378	77	M	C	None	None	Weight loss		3	Pneumonia

fibrous connective tissue. Variations in the structural arrangement of the cells, the amount and quality of the stroma, and the extent of secondary changes, such as hemorrhage and necrosis, were responsible for the differences in microscopic and gross appearances of the growths.

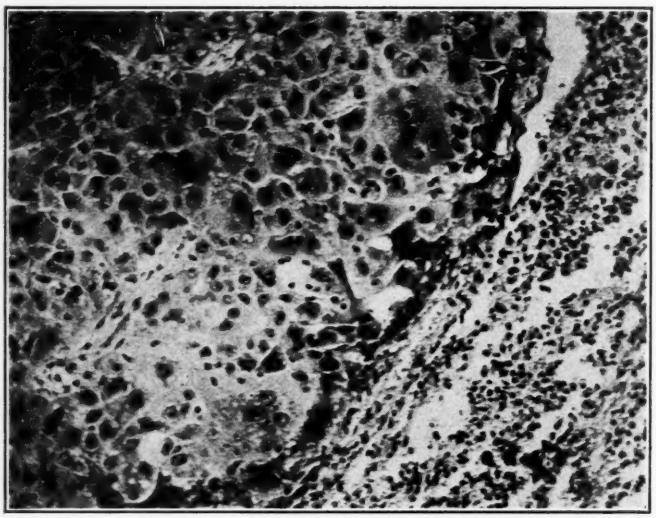

Fig. 1.—Metastatic liver cell carcinoma of the lung in a case of primary carcinoma of the liver (L-9).

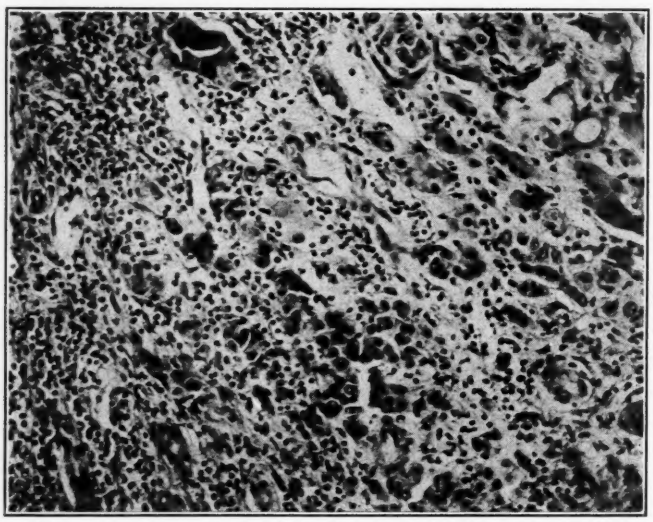

Fig. 2.—Carcinoma of the liver, liver cell type (L-10). Islands of liver cells are preserved amidst sheets of tumor cells.

The microscopic structure of the cylindrical cell carcinomas in this series was similar, whether the new growth was primary in the liver, the extrahepatic biliary ducts, or the gallbladder. Acinar or tubular structures lined by low cuboidal or tall columnar cells were embedded

in a newly formed loose connective tissue stroma which frequently replaced the underlying structures. Round or oval deeply staining vesicular nuclei were usually situated towards the base of the cells. Heaping up of the nuclei, budding of the epithelium, and cells in a state of

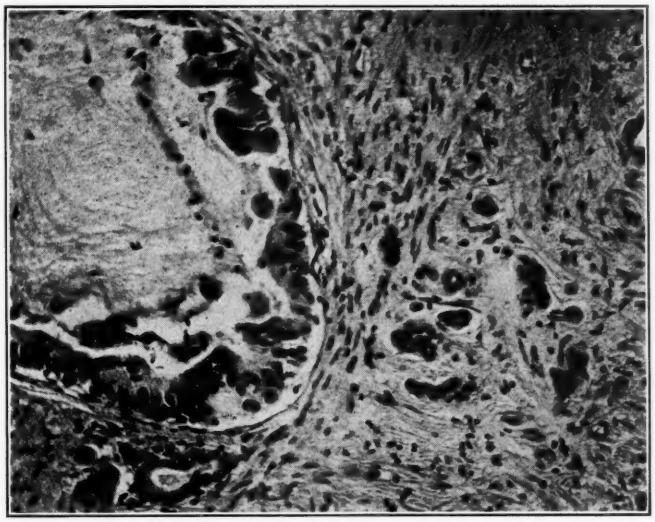

Fig. 3.—Carcinoma of the gallbladder, cylindrical cell type (G-3). Small islands of tumor cells are infiltrating tissue spaces in the muscular coat.

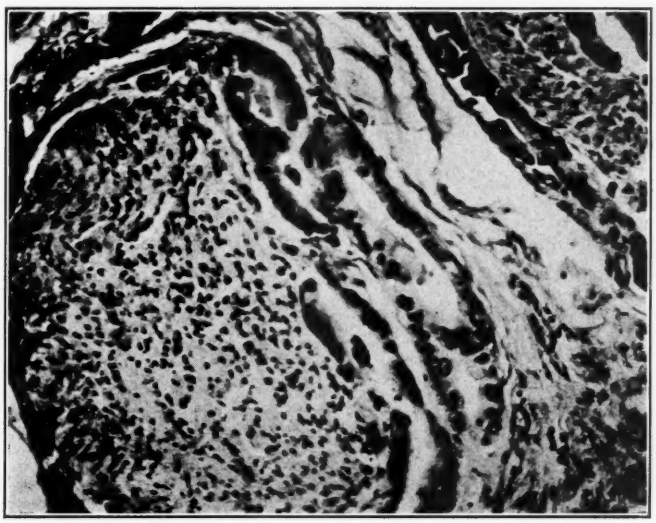

Fig. 4.—Carcinoma of the extrahepatic biliary ducts, cylindrical cell type (D-4). Extension along the sheaths of nerves is a conspicuous feature.

mitotic division were more frequent in some tumors than in others. Small islands or larger solid cords of tumor cells were numerous amidst the acinar and tubular structures. In the wall of the gallbladder small islands of tumor cells frequently infiltrated tissue spaces in the mus-

cular coat (Fig. 3). Along the margins of the growths, there were dense infiltrations of small round cells, large mononuclear cells, and polymorphonuclear leucocytes. Extensions along lymph channels and along the sheaths of nerves were conspicuous features of these tumors (Fig. 4). The amount of hemorrhage and necrosis varied according to the size of the growths.

SPREAD

In many instances, in addition to local infiltration or spread by lymph and blood channels, the growths metastasized to regional lymph nodes and distant organs.

In the liver cell carcinomas there was a single growth without metastasis in 2 cases (L-11 and L-13); extensive involvement of the liver in 4 cases; regional lymph node involvement in 6 cases; extensive peritoneal spread in 3 cases; lung involvement in 9 cases; and invasion of the suprarenals in 1 case.

In the 7 cases of primary cylindrical cell carcinomas of the liver, the regional lymph nodes and lungs were involved in 2; the lungs alone, in 1.

In the 6 cases of cylindrical cell carcinomas originating in the extrahepatic biliary ducts, there were extensions to the liver in 4 cases, with additional involvement of the regional lymph nodes in 1 case, the pancreas alone in 1 case, and the pancreas and regional lymph nodes in 1 case. Spread to the gallbladder occurred in 2 cases, in 1 of which the regional lymph nodes were also involved. Metastasis to the lungs occurred in 2 instances, the kidneys being additionally invaded in 1 of these.

In the 11 cases of cylindrical cell primary carcinomas of the gallbladder, no regional or distant spread was noted in 3. The regional lymph nodes alone were involved in 2; the liver alone, in 2; the regional lymph nodes and the liver, in 1; the regional lymph nodes, the liver, and the spleen, in 1; the liver and peritoneum, in 1; and the liver and lungs, in 1.

PREVIOUS HISTORY, CLINICAL MANIFESTATIONS, COURSE OF DISEASE

The majority of the patients with liver cell carcinomas were jaundiced and showed cirrhosis of the liver with ascites. Six of the 7 patients with primary cylindrical cell carcinomas of the liver had cirrhosis; 5 had ascites; 4 were jaundiced; and 1 had cholelithiasis.

All 6 patients with primary cylindrical cell carcinomas of the extrahepatic biliary ducts were jaundiced; 6 had cholelithiasis (mixed gallstones) and 1 had cirrhosis of the liver with ascites.

Four of the 11 patients with primary cylindrical cell carcinomas of the gallbladder had cholelithiasis, jaundice, and cirrhosis of the liver

with ascites; 1 had cholelithiasis, jaundice, and cirrhosis; 1 had cholelithiasis and ascites; 1, cholelithiasis and cirrhosis; 1, jaundice and cirrhosis; 1, cirrhosis only.

The temperature of all the patients, irrespective of the type or location of the tumor, showed only slight variations from the normal. In 15, however, rise of temperature occurred following operation or intercurrent diseases (nephritis, pneumonia, etc.). There was no significant discrepancy between the temperature and the rate of respiration. Although the majority of the patients were jaundiced, their pulse rates were usually slightly elevated. A clinical diagnosis of syphilis was recorded in 6 of the 40 patients (L-2, L-4, L-11, L-13, C-3, G-6).

Six of the patients with liver cell carcinomas (L-3, L-4, L-5, L-9, L-15, L-16) were moderate or heavy drinkers; 2 of those showing cylindrical celled tumors (G-1 and G-2) gave histories of overindulgence in alcoholic beverages.

The illnesses lasted for two to sixteen months, with an average duration of four and one-half months.

CAUSE OF DEATH

In all but 2 of the patients in whom the neoplasms were incidental observations, the new growth was the principal lesion, and in 25 patients was the immediate cause of death.

COMMENT

The older reports of Wells,[1] Wheeler,[2] Goldzieher and Bókay,[3] Yamagiwa,[4] Karsner,[5] Winternitz,[6] and the more recent ones of Fried,[7] Rowen and Mallory,[8] Counseller and McIndoe,[9] Strong and Pitts,[10] Tull,[11] Brines,[12] and others on neoplasms of the biliary system show that the incidence of primary carcinomas of the liver varies in different geographic locations. Similar variation seems to exist as regards the site of the primary growth. Our series is remarkable in that the primary carcinomas of the liver have a high incidence and far outnumber those primary in the extrahepatic biliary ducts and in the gallbladder.

The primary liver cell carcinomas, in accord with the statistics compiled by others, outnumbered the cylindrical cell carcinomas. In our series the structure of the cylindrical cell carcinomas occurring primary in the liver was identical with that of those arising from the extrahepatic biliary ducts and the gallbladder.

Our series is furthermore unusual in that practically all of the patients with primary liver carcinomas had cirrhosis of the liver with ascites, irrespective of the histologic structure of the growths. Few of them had cholelithiasis; whereas, most of the patients with primary growths in the extrahepatic biliary ducts and the gallbladder had cholelithiasis. Jaundice occurred in practically all the cases, irrespective of the site or histologic type of the primary growth.

SUMMARY

In 6,050 necropsies performed on persons over one year of age, there were 40 primary carcinomas of the biliary system. Of these, 23 were primary in the liver (16 liver cell and 7 cylindrical cell carcinomas); 6 were primary in the extrahepatic biliary ducts (all cylindrical cell carcinomas); 11 occurred primarily in the gallbladder (all cylindrical cell carcinomas).

Eight of the patients died in the fifth, 11 in the sixth, and 14 in the seventh decade of life. The youngest patient was thirteen years of age and the oldest was seventy-seven years of age.

In many instances, in addition to local infiltration or spread by lymph and blood channels, metastases occurred in regional lymph nodes or distant organs.

Practically all of the patients with primary liver growths had cirrhosis of the liver with ascites.

Most of the patients with primary growths in the extrahepatic biliary ducts and the gallbladder had cholelithiasis.

With few exceptions all of the patients were jaundiced.

The average duration of illness was four and one-half months.

REFERENCES

1. Wells, H. Gideon: Primary Carcinoma of the Liver, Am. J. M. Sc. **126**: 403-417, 1903.
2. Wheeler, F. J.: Some Observations on Primary Carcinoma of the Liver, Guy's Hosp. Rep. **48**: 225-244, 1909.
3. Goldzieher, Max, and v. Bókay, Zoltán: Der primäre Leberkrebs, Virchows Arch. f. path. Anat. **203**: 75-131, 1911.
4. Yamagiwa, K.: Zur Kenntnis des primären parenchymatösen Leberkarcinoms ("Hepatoma"), Virchows Arch. f. path. Anat. **206**: 437-467, 1911.
5. Karsner, Howard T.: A Clinicopathological Study of Primary Carcinoma of the Liver, Arch. Int. Med. **8**: 238-261, 1911.
6. Winternitz, M. C.: Primary Carcinoma of the Liver, Johns Hopkins Hosp. Rep. **17**: 143-184, 1916.
7. Fried, B. M.: Primary Carcinoma of the Liver, Am. J. M. Sc. **168**: 241-267, 1924.
8. Rowen, Henry S., and Mallory, F. B.: A Multinucleated Liver Cell Carcinoma, Am. J. Path. **1**: 677-680, 1925.
9. Counseller, Virgil S., and McIndoe, Archibald H.: Primary Carcinoma of the Liver, Arch. Int. Med. **37**: 363-387, 1926.
10. Strong, G. F., and Pitts, H. H.: Primary Carcinoma of the Liver, Arch. Int. Med. **46**: 105-120, 1930.
11. Tull, J. C.: Primary Carcinoma of the Liver: A Study of One Hundred and Thirty-Four Cases, J. Path. & Bact. **35**: 557-562, 1932.
12. Brines, Osborne Allen: Primary Tumors of the Liver, Am. J. Clin. Path. **3**: 221-235, 1933.

EXPERIMENTAL STUDY OF THE BEHAVIOR OF FREE FAT TRANSPLANTS*

CHARLES E. GURNEY, M.D.,† ROCHESTER, MINN.
(From the Mayo Foundation)

THE autotransplantation of detached pieces of fat is an established procedure in the field of plastic and reconstructive surgery. The fat grafts are used to obliterate dead spaces, to fill small depressions in the soft tissue for the purpose of restoring contour and mobility, to elevate depressed and adherent scars, and to prevent subsequent adhesions and contractures.

Fat grafts are also of value in general surgery where they have been employed in arthroplasty, epilepsy, tenorrhaphy, neurorrhaphy, and in certain pulmonary diseases. They have been used to fill cavities in bone and as a hemostatic agent in the operations on parenchymatous organs.

There are certain clinical problems associated with the transplantation of fat which justify further experimental study. It is well known that practically all fat grafts gradually diminish in size following their transplantation. The surgeon attempts to compensate for this shrinkage by transplanting a larger piece of tissue than would at first seem necessary. The amount of shrinkage may vary widely with different grafts. Since this cannot be accurately determined beforehand, the amount of fatty tissue which should be transplanted can only be roughly estimated. This rather indefinite method of overcorrecting for an indeterminate amount of shrinkage accounts for some of the unsatisfactory final results. Occasionally, one encounters a partial or complete liquefaction of the graft without any apparent infection.

As regards technique, there arises the question of whether multiple small pieces survive better than does a single piece of equal bulk, whether fat from one site is preferable to that from another, and whether one can successfully transplant fat from one individual to another. Of academic interest is the question of whether the graft remains as original fat, whether it is replaced by new fat from the host, or whether it is entirely replaced by scar tissue. The present experimental study was designed to answer, in part, some of these problems.

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†Fellow in Oral and Plastic Surgery, the Mayo Foundation,

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REVIEW OF THE LITERATURE

The first clinical attempt at the transplantation of fat was made by Neuber in 1893. He reported the successful use of small free grafts of fat in cosmetic operations about the orbit and stressed the use of small grafts. His large grafts always resulted in failure. He also reported experiments with fat implants to fill cavities in bone, but only to condemn their use. Three years later (1896) Silex claimed good cosmetic results following the use of fat transplants in the treatment of depressed scars about the orbit. In this same year Czerny reported the successful transplantation of a large lipoma to replace a breast which had been removed because of chronic cystic mastitis. Verderame (1909) reported ten years' experience with fat transplantation in ocular surgery. He recognized that the fat graft shrank considerably and advocated the use of larger transplants than appeared necessary to fill the defect. Since that time, numerous reports of the successful use of fat transplants in a wide variety of conditions have appeared in the surgical literature.

There have been few experimental studies designed to determine the fate of free fat transplants. The most recent work is that of Loeb (1930), who transplanted fat in connection with cartilage. He expressed the opinion that after autotransplantation certain tissues may remain alive permanently, as, for instance, thyroid gland, fat tissue, and cartilage, but after homotransplantation the majority of these tissues are preserved only for a short time.

Davis and Traut maintained that the histologic changes which occur in transplants of fat closely approximate those which occur in transplanted bone. They also were of the opinion that none of the transplanted fat actually survives but after a primary degeneration is replaced by embryonic type of cells which later develop into fat cells or are replaced by fibrous connective tissue.

With regard to omental fat, Mann (1921) said that a free transplant of omentum in the peritoneal cavity may remain seemingly viable for as long as one year, and retain some, although only a small percentage, of its fat. In many instances, however, after a few weeks he observed that the transplants were reduced to an almost fat-free scarlike tissue.

Carl B. Davis (1917) experimented with free transplantation of omentum both subcutaneously and within the abdomen. He concluded that omentum, transplanted freely beneath the skin in masses 1 inch in diameter, maintains the greater portion of its bulk. His microscopic examination of the grafts showed an astonishing viability. It seemed to be of little consequence whether the graft was ½ inch (1.27 cm.) or 3 inches (7.6 cm.) in diameter.

Lexer has had a wide experience in the transplantation of tissues generally. In regard to fat, he said that fatty cysts occur in the cen-

tral part of the grafts. He contended that about one-third of the fat graft survives and that the graft does not grow in proportion to the individual. He called attention to the fact that multiple pieces in a transplant do not survive as well as does a single piece, "because they are exposed to more danger and because they are more likely to become overgrown with connective tissue."

Histologic studies on free fat transplants have been made by Hilse (1928) who concluded that in all free transplantation of fatty tissue, which was carried out under normal conditions, there is a regeneration of fatty tissue without any exception. He referred to the histiocyte filled with fat as a "lipoblast."

SCOPE OF PRESENT STUDY

The present study consisted of making autografts and homografts of fat in 185 male and female white rats of average size. In each instance, except as mentioned later, the following technique was used. Under ether anesthesia, the subcutaneous fat in the right groin was removed through a small incision. Although the individual pieces were not measured at the time of transplantation, those of a control series were measured as a basis for future comparison. The average size of the grafts was found to be 1,705 cu. mm., about half the size of the distal phalanx of the index finger. In transplanting the grafts, a small incision was made through the skin over the ventral aspect of the thorax on the left side. By inserting the points of the scissors and gently separating them, a subcutaneous pocket was made in the connective tissue into which the fat graft was placed. The thoracic region was chosen to receive the graft because, except for a thin layer, it is relatively free from adipose tissue, which enables the detection of even small amounts of foreign fat. Both incisions were closed with suture.

In the rat an irregular piece of fat about 40 to 60 mm. long by 5 to 8 mm. in diameter lies free within the abdomen except for its narrow attachment to the testes and epididymis. It is completely surrounded by peritoneum. In twenty rats, this peritonealized testicular fat was substituted for the fat of the groin. The average size of these grafts at the time of transplantation was found to be 1,550 cu. mm.

For convenience, the rats were divided into five groups, determined by the treatment which the fat received immediately preceding its transplantation. The number of rats in each group represents the number which survived. In group A (32 rats), autotransplants of fat of the groin were transferred without trauma. In group B (30 rats), autotransplants of fat of the groin were cut into about ten equal pieces with a sharp razor; all pieces were placed together in the thoracic area. In group C (31 rats), autotransplants of fat of the groin were crushed severely with a hemostat immediately before they were transplanted. In group D (30 rats), homotransplants of fat of the groin were trans-

planted as a single piece without trauma. In group E (13 rats), autografts of peritonealized testicular fat from the abdominal cavity were transplanted as a single piece without trauma.

In the first four groups, the rats were killed at intervals of one, two, three weeks and one, two, three, four, six, nine, and twelve months after operation. In group E examinations were made at one week and one, four, nine, and twelve months after operation. In most instances 3 animals were examined at each of these intervals, but in several cases 4 and even 5 were studied. Although a sufficient number of rats were operated on to permit a 25 per cent loss, in two instances the loss exceeded this amount and only 2 rats remained for examination. The surviving grafts were measured and their size computed in terms of

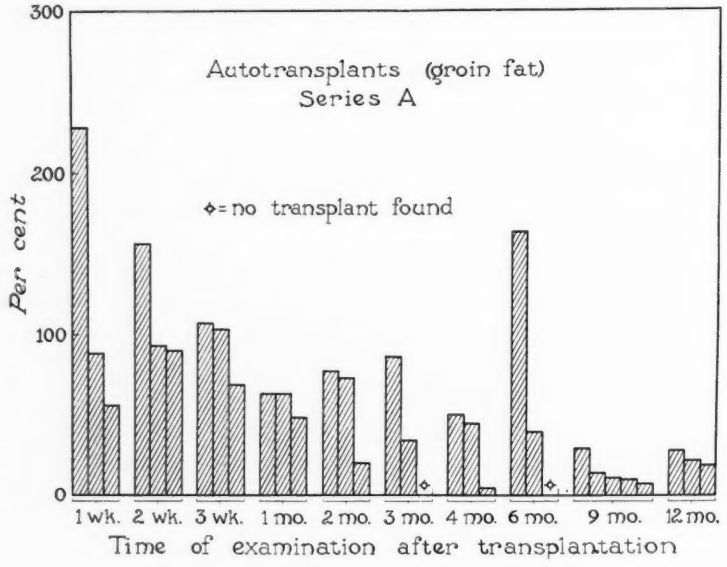

Fig. 1.—Size of transplants in group A.

cubic millimeters for comparison with their original size. They were then studied both grossly and microscopically. All of the sections were stained with sudan III, and in some specimens this was supplemented with hematoxylin and eosin.

EXPERIMENTAL OBSERVATIONS

Gross Appearance.—The outstanding gross finding was that almost without exception those autografts which were transplanted as a single piece without trauma (groups A and E) retained their identity throughout the whole year of observation (Figs. 1 and 2). In these figures and in subsequent figures which show the size of the grafts at various intervals after operation, the size of the graft is expressed as the percentage of the size of the graft at the time of transplantation. Each

column represents one animal. Those autografts which were cut into multiple pieces or crushed (groups B and C) survived for a shorter period. Except for one small graft in group C which survived for a year, and one in group B which survived for six months, the transplants in both groups had completely disappeared after four months (Figs. 3 and 4). The homografts (group D) survived only for a very short time. Not the slightest suggestion of a graft was found after three months (Fig. 5). In those instances in which the graft had disappeared, an attempt was made to cut sections at the site of the implant, but with few exceptions the graft had disappeared so completely that no abnormality remained in the tissues to suggest its former situation.

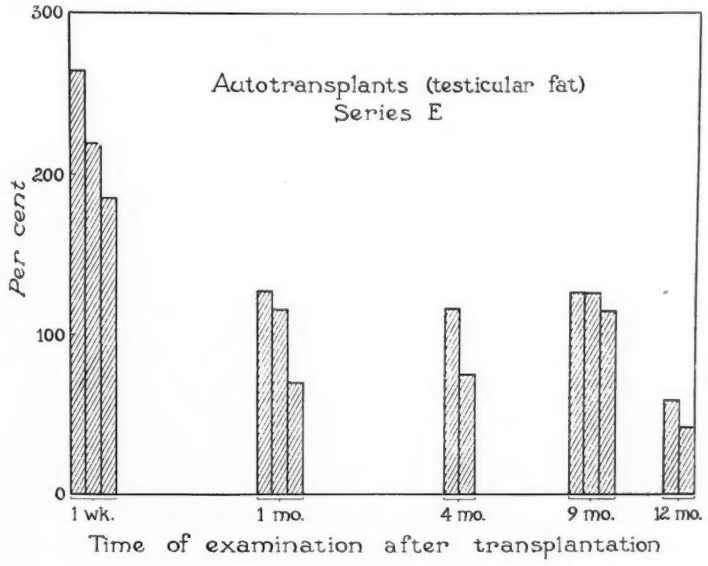

Fig. 2.—Size of transplants in group E.

All of the grafts were soft to palpation for the first day or two after operation. About the third or fourth day they began to enlarge and became more firm and globular. This progressed for about a week. By the second week they began to recede, and by one month they were soft and much smaller than they were when transplanted. Gross infection occurred in seven cases and in all except one case the infection occurred in those grafts which had been subjected to cutting or crushing. The infected grafts were soft, dark colored, and apparently had completely disintegrated. Some contained a definite purulent material while others were filled with a fine granular cheesy substance. In several cases the graft had partially or wholly degenerated apparently because of an associated hematoma. One degenerated graft was separated from the host by a thin layer of old blood which completely

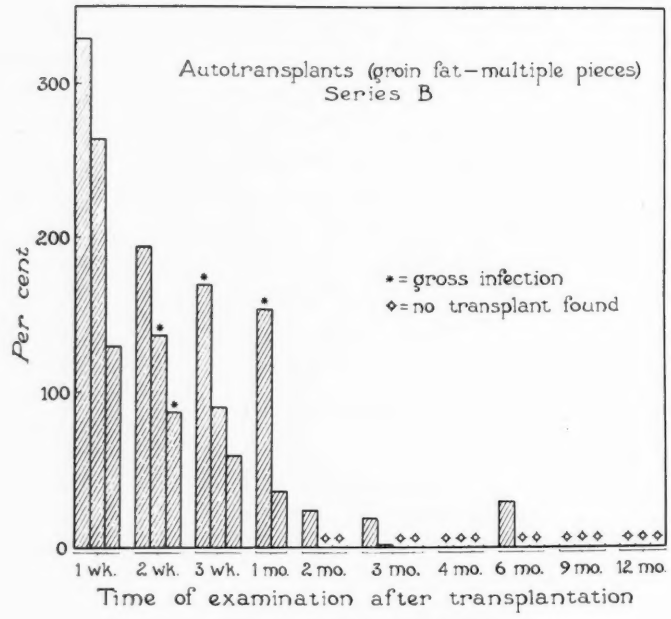

Fig. 3.—Size of transplants in group B.

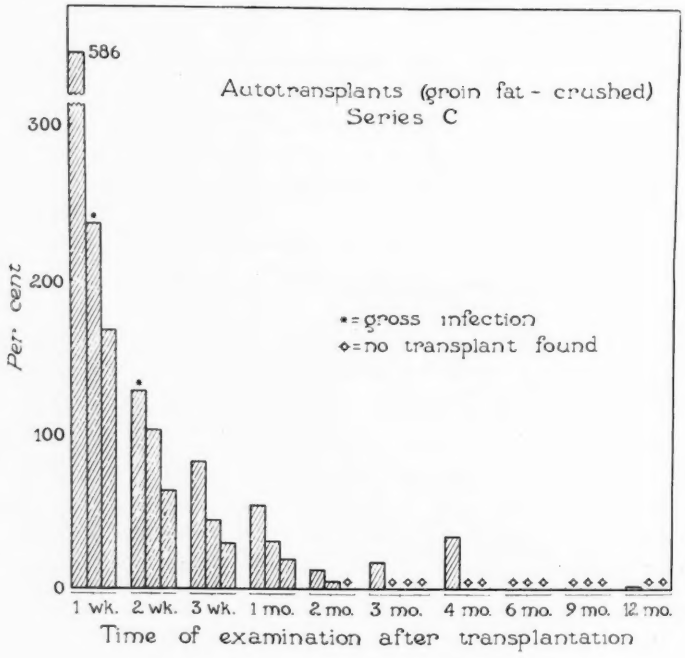

Fig. 4.—Size of transplants in group C.

surrounded it. The cut surface of most of the grafts presented a gray homogenous appearance not unlike that of normal fat. The grafts composed of multiple pieces and those which had been subjected to crushing were much more oily. Minute droplets of fat exuded from the cut surface of these grafts. There was no complete liquefaction of the graft as occasionally is seen clinically.

It is of interest to note that the region in the right groin, from which the implant was taken, was not refilled with fat in any of the specimens. Nature apparently does not attempt to restore lost fatty tissue.

Microscopic Appearance.—The microscopic findings were characterized by degenerative changes in all of the grafts. The chief difference was one of degree and extent of involvement. The most violent and extensive reaction was seen in the homotransplants (group D). Those

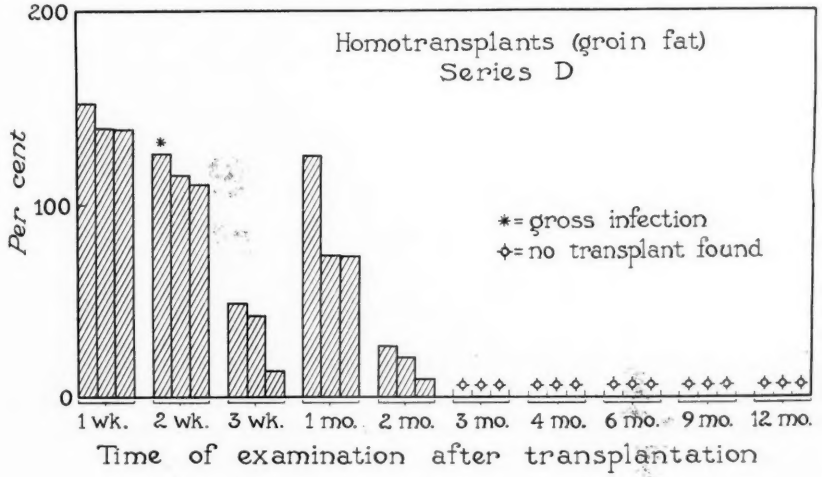

Fig. 5.—Size of transplants in group D.

grafts composed of multiple pieces and those which had been crushed (groups B and C) were subjected to a less violent reaction, while those grafts which had been carefully transplanted as a single piece without trauma showed the least degenerative change (Fig. 6*a*, *b*, and *c*).

The degenerative changes were manifested by a disappearance of the nucleus of the fat cell; a disintegration of the wall of the cell, and the formation of cysts and vacuoles, infiltration of polymorphonuclear leucocytes, lymphocytes, and histiocytes; phagocytosis, and a gradual reduction in the size of the graft, which in some instances continued until the graft entirely disappeared.

The fat cells in certain portions of the graft appeared normal except for the absence of their nuclei. This finding was most noticeable in the homografts one week after operation; this change involved most of these grafts (Fig. 7*a*). In the other groups it occurred only in

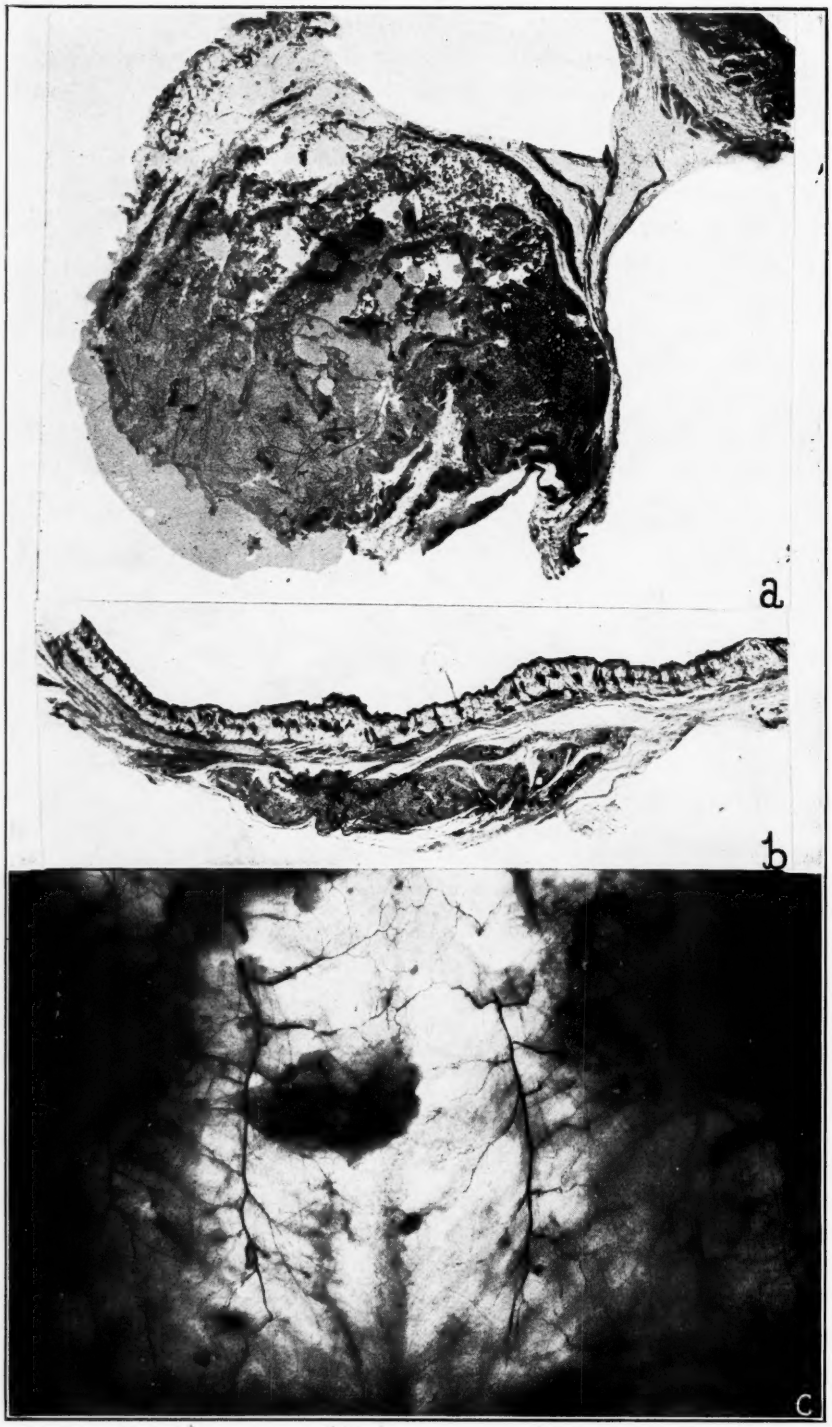

Fig. 6.—Grafts in group A; *a*, one week after transplantation, small portion of fat is well preserved but the rest of the graft is undergoing degeneration (×5½); *b*, nine months after transplantation, the graft is about a fourth of its original size but almost all of the graft appears normal (×5½); *c*, graft in group A, nine months after transplantation; appearance of gross specimen attached to skin, as seen by transmitted light (×260).

portions of the grafts, especially about the periphery. The disintegration of the wall of the fat cell apparently resulted in the formation of small accumulations of fat, which appeared as fatty cysts within the transplant. Associated with these were large and numerous spaces which appeared as vacuoles. Both the cysts and vacuoles were found chiefly at the periphery of the graft, except in the homografts, where they occurred throughout the whole transplant. Polymorphonuclear leucocytes were observed in almost all the grafts but rarely after the first few weeks. Many of the leucocytes were filled with fat. Lymphocytes were present in small numbers. The histiocytes were numerous

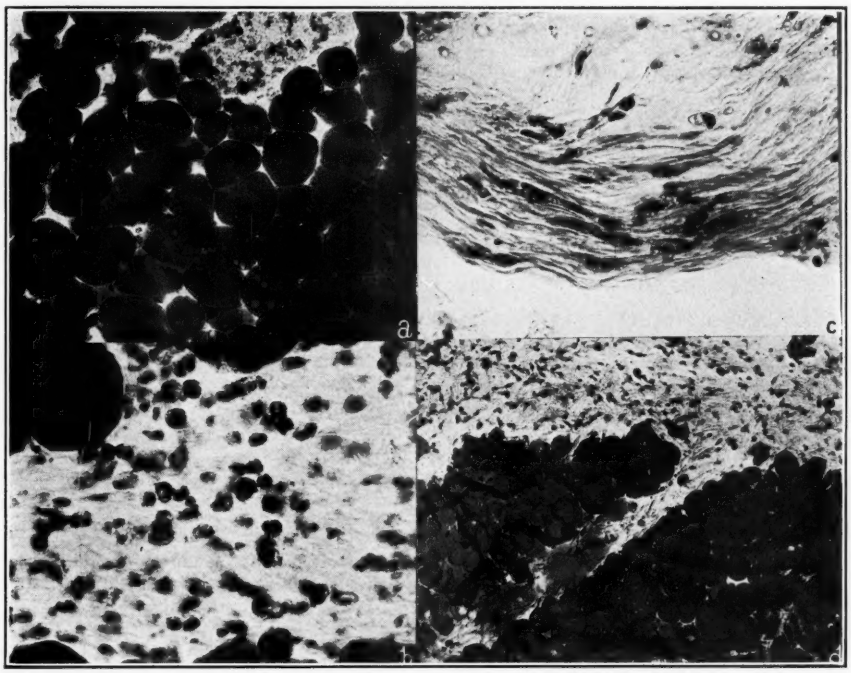

Fig. 7.—*a*, Graft (homograft) in group D, one week after transplantation; fat cells appear normal except that they do not have any nuclei (×400); *b*, graft in group A, two weeks after transplantation; connective tissue contains histiocytes and polymorphonuclear leucocytes which are filled with globules of fat (×400); *c*, graft in group A, three weeks after transplantation; connective tissue wall at periphery showing numerous fibroblasts filled with fat (×260); *d*, graft in group A, one week after transplantation; many new blood vessels are present in the fat (×365).

in most of the sections and appeared to be the chief phagocytic cell (Fig. 7*b*). Fibroblasts filled with particles of fat were common, especially after the first few weeks. They were found in the new connective tissue about the periphery and in the septa within the graft (Fig. 7*c*). No giant cells were found in any of the grafts.

A definite, but futile, search was made to find new fat cells which would lend support to the assertion that regeneration of fat is the rule after transplantation. All of the fat appearing in the grafts as viable tissue was composed of normal adult fat cells. New blood vessels were

visible as early as one week in the autografts which had been made without trauma (Fig. 7*d*). They could be followed at succeeding examinations and at nine and twelve months the grafts were well vascularized (Fig. 6*b*). No new vessels were observed in any of the homografts and rarely in the autografts which had been subjected to trauma. There apparently had been an attempt to wall off the transplanted tissue, because as early as one week, a definite compact layer of cells could be found surrounding the graft. It was most marked in the homografts and least marked in those autografts which survived.

It was apparent that the degenerating portion of each graft was constantly being removed from the site of the implant, while the remainder became vascularized early and continued as normal fat. This accounted for the finding of a gradual increase in the percentage of normal appearing fat in the grafts throughout the year. During the first month, only a small percentage of the implant contained normal fat, but by one year about 90 per cent of the graft appeared normal.

COMMENT

It seems justifiable to assume that, in those portions of the graft which did not survive, the vitality of the cells had been jeopardized in some manner, as evidenced by the disappearance of their nuclei. Since the nucleus is the vital part of the cell, it follows that the breakdown of the wall of the cell was only a natural sequence. This liberation of fat by contiguous cells probably gave rise to the fatty cysts. If a few fat cells only became involved, a small fatty cyst resulted, which, in some cases, persisted in the sections. If, on the other hand, many fat cells broke down, the resulting large cyst would likely lose its contents, during the technique incident to staining, and thus would appear in the sections as a vacuole. It therefore seems apparent that the fatty cysts, which sometimes appeared as vacuoles, were formed by the coalescence of the adjacent fat cells, the walls of which had disintegrated. Phagocytosis of this liberated fat was accomplished chiefly by the histiocytes assisted by the polymorphonuclear leucocytes. Opinions differ regarding the rôle of the fibroblast as a phagocytic cell. It is generally agreed that the histiocyte may be transformed into the fibroblast, and that the phagocytic properties of the latter are negligible when compared to those of the histiocyte. Nevertheless, that portion of any graft which showed evidence of degeneration was ultimately disposed of by the host and only a small viable portion was left. In the group homografts, not even a small portion of the graft remained.

It was shown that the percentage of normal fat in any surviving graft gradually increased throughout the year. This observation does not justify the assumption that new fat was being produced. A more

rational explanation is that a certain portion of the transplanted tissue gained an adequate blood supply early and continued to survive, while the remainder of the graft degenerated and was gradually eliminated from the site of the implant. If new fat had been formed, one would expect to find new fat cells at some of the examinations during the year. No such new fat cells were found. It therefore seems justifiable to conclude, from these studies, that the fat seen at the site of transplant, one year after transplantation, was a portion of the original fat.

The complete disappearance of the homotransplants needs no further comment, other than it conformed to the accepted opinion regarding homotransplants in general. They are notorious for their inability to survive definitely, although Loeb has shown that their survival is more likely to be prolonged if a closer relationship exists between donor and host.

The eventual disappearance of the crushed grafts and those composed of multiple pieces attests the devastating effect of trauma on the vitality of a graft. This is probably the reason why the grafts which were composed of peritonealized fat retained a larger proportion of their original tissue than did those grafts composed of fat from the groin. In the latter case all of the cells at the periphery of the graft were subjected to the injury of cutting when the fat was being removed for transplantation. The finger-like projection of abdominal fat was surrounded by a protective layer of peritoneum, except at its attachment to the testis, and could therefore be removed with relatively slight injury to its cells.

The assertion has been made that those fat transplants which do not survive are converted into scar tissue. The present findings with subcutaneous fat do not substantiate this view. In those groups in which the grafts had disappeared completely, there was insufficient thickening of the tissues even to locate the site of the implant. The dense layer of fibrous tissue which forms around the homotransplants early also disappeared with the disappearance of the graft itself. If the degenerated portion of the surviving grafts had been converted into scar tissue, one would expect to find this tissue around the grafts in the later stages. No such scar tissue was found.

These experiments do not justify the opinion that multiple small pieces of fat survive better than does a single large piece. They show that single pieces remain viable for at least one year, while original grafts of a similar size, and composed of several smaller pieces of fat, may last as long as six months, but the majority disappear by the third month. The experiments also confirm Lexer's contention that the fat grafts are ultimately about a third the size that they were at the time of transplantation. The transplants which were obtained from

the groin averaged 23 per cent of their original size and those which were obtained from the abdominal cavity 51 per cent of their original size.

These experiments have given only a slight suggestion regarding the mechanism of liquefaction of a fat graft. No complete liquefaction was observed in any of the specimens. The nearest approach to it was found in some of those grafts which had been cut or crushed. Their cut surface exuded a thin oily fluid which was thought to be fat. This fat most likely came from the fatty cysts which were formed by the coalescence of adjacent fat cells, whose walls had disintegrated. Since this gross evidence of liquid fat did not occur in the nontraumatized autotransplants, it suggests that trauma may be a definite causative factor of liquefaction.

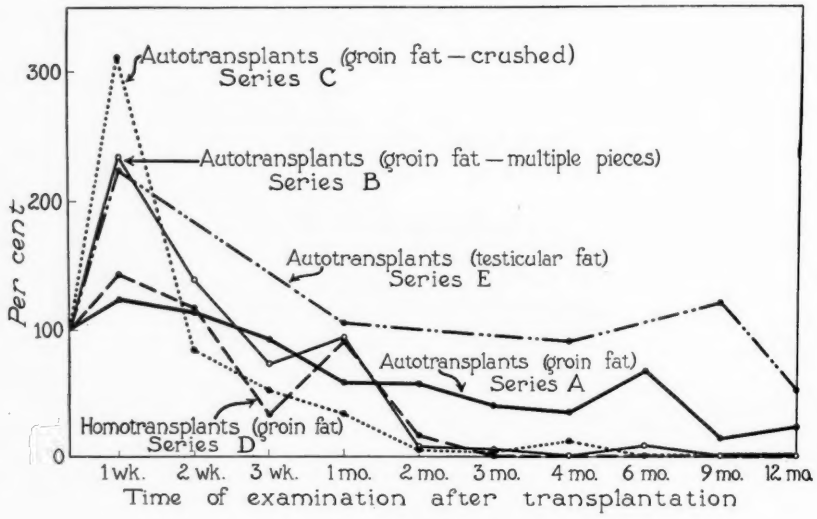

Fig. 8.—Size of transplants in different groups.

SUMMARY

Experimental studies designed to show the behavior of free transplants of fat were made on 185 white adult rats of average size. In each instance subcutaneous or peritoneal fat was transferred to a subcutaneous pocket in the thoracic region. The rats were divided into five groups, depending on the treatment received by the graft immediately prior to its transplantation. Group A comprised the rats in which autotransplants of fat of the groin were transferred as a single piece without trauma. Group B included those in which autotransplants of fat of the groin were cut into multiple pieces with a sharp razor. Group C included those in which autotransplants of fat of the groin were severely traumatized by squeezing with a hemostat. Group D comprised those in which homotransplants of fat of the groin were

transferred without trauma. Group E included those in which autotransplants of fat obtained from the peritoneal cavity were transferred without trauma.

The rats in the first four groups were killed at intervals of one, two, and three weeks and one, two, three, four, six, nine, and twelve months after the operation. In the last group they were killed at one week and one, four, nine, and twelve months after the operation. At each examination the surviving grafts were studied grossly and microscopically. All of the sections were stained with sudan III and a few were stained with hematoxylin and eosin.

The homotransplants survived only for a short time; none was found after the third month. The crushed grafts and those composed of multiple pieces survived a little longer. The autografts of subcutaneous and peritoneal fat, which were transferred without trauma, persisted for a year although they were from one-third to one-half of their original size (Fig. 8).

CONCLUSIONS

This experimental study would seem to justify the following conclusions:

1. Subcutaneous fat is a suitable tissue for autotransplantation if proper precautions are taken at the time of transplantation.

2. The transplantation of multiple pieces of fat, in lieu of a single piece of equal bulk, is not a satisfactory procedure.

3. Care should be exercised in handling the tissue to be grafted, as trauma definitely lessens its ability to survive, in addition to inviting gross infection.

4. Absolute hemostasis is essential to the success of transplantation of fat, since even a slight hemorrhage in the wound greatly jeopardizes the viability of the graft.

5. Although peritoneal fat survives better than does subcutaneous fat, the accessibility and success of the latter give it preference in the average case.

6. A much larger piece of fat must be transplanted than would at first seem necessary, since only one-fourth to one-half of the graft survives.

7. The fat comprising the graft at one year after operation is a portion of the original transplanted fat.

8. The fat in that portion of the graft which does not survive is phagocytosed chiefly by the histiocytes and secondarily by the polymorphonuclear leucocytes. This accounts for the reduction in the size of the graft.

9. If that portion of the fat which disappears does become scar tissue, this also ultimately disappears, since the site of the transplant is not marked grossly by scar tissue.

10. Phagocytic cells do not use their fat to form new fat cells during the first year after transplantation.

11. Absolute asepsis is necessary to successful transplantation of fat. Although slight infection results in only a small loss of tissue, which the graft can hardly afford, gross infection leads to a loss of the entire graft.

12. The original fat which is removed for transplantation does not become replaced during the first year after such removal.

REFERENCES

1. Czerny: Quoted by Neuhof, Harold: The Transplantation of Tissues. New York, 1923, D. Appleton-Century Co., Inc., pp. 70-91.
2. Davis, C. B.: Free Transplantation of the Omentum, Subcutaneously and Within the Abdomen, J. A. M. A. **68**: 705, 706, 1917.
3. Davis, J. S., and Traut, H. F.: Transplantation of Fat, in Lewis, Dean: Practice of Surgery, Hagerstown, Maryland, F. W. Prior Company, Inc., vol. 5, chap. 8, pp. 98-124.
4. Hilse, A.: Histologische Ergebnisse der experimentellen freien Fettgewebstransplantation, Beitr. z. path. Anat. u. z. allg. Path. **79**: 592-624, 1928.
5. Lexer, Erich: 20 Jahre Transplantationsforschung in der Chirurgie, Arch. f. klin. Chir. **138**: 251-302, 1925.
6. Loeb, Leo: Transplantation and Individuality. Physiol. Rev. **10**: 547-616, 1930.
7. Mann, F. C.: The Transplantation of Fat in the Peritoneal Cavity, Surg. Clin. North America **1**: 1465-1471, 1921.
8. Neuber: Quoted by Neuhof, Harold: The Transplantation of Tissues, New York, 1923, D. Appleton-Century Co., Inc., pp. 70-91.
9. Silex: Quoted by Neuhof, Harold: The Transplantation of Tissues, New York, 1923, D. Appleton-Century Co., Inc., pp. 70-91.
10. Verderame: Quoted by Neuhof, Harold: The Transplantation of Tissues, New York, 1923, D. Appleton-Century Co., Inc., pp. 70-91.

A METHOD OF CONSTANT SUCTION APPLIED TO THE LEVINE TUBE

CLAUDE E. WELCH, M.D., BOSTON, MASS.
(From the Surgical Service, Massachusetts General Hospital)

THE advantages of some form of drainage of the upper intestinal tract by means of the Levine tube have been appreciated for many years. Such drainage may be secured in any of several ways. The catheter either may be introduced into the stomach, the stomach may be emptied and the tube withdrawn, or the tube may be left in position draining by gravity, or some form of continuous suction may be applied. Each of these methods has its indications and its limitations.

It is to Wangensteen[1] that the greatest credit must be given for the popularization of the application of continuous suction to the gastrointestinal tract. The essential feature of his apparatus is that a continuous suction force of about 75 cm. of water is applied either to the stomach, or, preferably, to the duodenum. Stronger suction must be avoided because intestinal mucosa may be traumatized by being drawn into the tube. On the other hand, if the suction is much less than 75 cm. of water, the stomach will not be thoroughly emptied.

There are certain disadvantages of the original Wangensteen apparatus. The fluid in the various tubes often falls because of loose connections, or the water column becomes broken frequently by the withdrawal of gas from the stomach so that the siphon is broken. Consequently, careful attention must be given each unit. Granted that perfect glassware and tubing are present—often a phenomenon in a large hospital—the nursing staff must be continually on guard to change bottles and must have a great deal of intelligence to keep the tubes clear. Suffice it to say that we finally became inured to the sight of the apparatus working less than 75 per cent of the time, although frequently it has required the entire time of a nurse to keep changing the bottles on a single ward.

In other words, the original Wangensteen apparatus has proved unsatisfactory in our wards. For that reason we have recently employed an apparatus which has the great advantage of simplicity and is certain to function continuously. A somewhat similar apparatus has been used in many clinics in the treatment of empyema or other pleural effusions. Iselin,[2] Carlson,[3] Wangensteen,[4] and Churchill, among others, have used such a device following chest surgery.

This method requires continual suction such as is now found in most hospitals. The apparatus may also be attached to a water or an

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electric suction pump. It is diagrammed in Fig. 1. An ordinary Levine tube (*A*) with several holes in the tip is passed into the stomach or duodenum. The tube is then connected to a 2½-liter bottle (*B*), which serves as a collecting bottle. It is important that there should not be long loops in the connection between the nose and the bottle if the full amount of suction is to be maintained. The bottle *B* is connected by a short rubber tube to a trap *C*, which is joined to the mercury valve *D*, which consists of a glass cylinder of small diameter about 20 cm. high. An ordinary 500-c.c. bottle may be used, but the smaller the diameter of the bottle the less mercury will be needed. The cylinder should be tall in order to reduce the amount of spattering of the mercury. The tube *E* is an open glass tube extending 5.5 to 6 cm. below the level of the mercury. The bore of this tube should be about 2 mm.; a larger tube is more noisy in operation. The mercury valve is connected to a trap *G* by means of a short rubber tube *F*. The empty cylinders *C* and

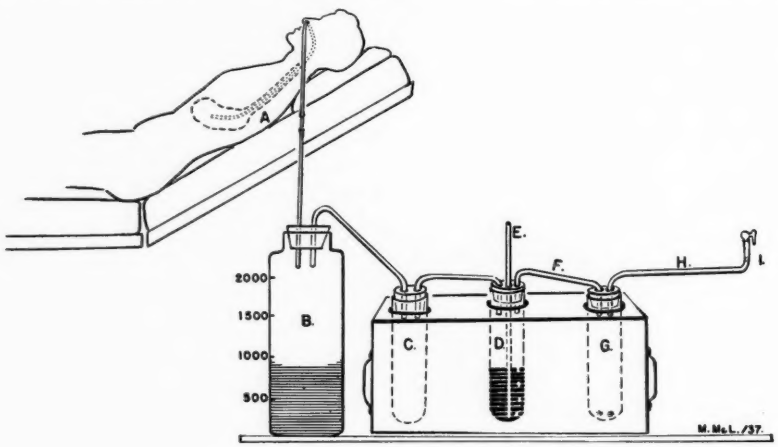

Fig. 1.

G are similar in size to *D*. Any mercury that should escape from *D* is caught by *G* which is connected by another rubber tube *H* to the source of constant suction, *I*.

In order to operate the tube, suction is turned on until bubbles of air are drawn through the tube *E* and escape through the mercury. Only an occasional bubble must escape to keep the suction constant at 75 cm. of water; there is no need to keep the mercury bubbling as loudly as possible. The two traps *C* and *G* allow either end of the apparatus to be connected to the suction and the other to the nasal tube. It thus becomes impossible to connect the apparatus in the wrong direction. As the collecting bottle, *B*, fills, it is measured and emptied. This rarely will be necessary more often than once in twenty-four hours. The original Wangensteen apparatus required attention very frequently because so much gas is removed from an obstructed intestine. In this arrangement gas is carried away by the suction.

The mercury valve must be protected from breakage. A satisfactory method is to obtain a wooden block about 30 by 20 by 20 cm. in size. Three holes are then bored in the block to receive the mercury valve and the traps. A little cotton should be placed beneath each tube. If breakage should occur, the mercury may be swept up from the floor, filtered, and used again. The cost of the apparatus therefore is not excessive. About 2 pounds of mercury will be required if the valve is 2.5 cm. in diameter. The valve will become cloudy gradually because of deposition of a thin layer of mercury on the glass wall necessitating an occasional cleaning of the glassware. This portable apparatus, therefore, is not expensive, and, since in addition it is almost unbreakable and requires little intelligence to place it in operation, it possesses the most important features of any useful hospital equipment.

REFERENCES

1. Wangensteen, O. H., and Paine, J. R.: Treatment of Acute Intestinal Obstruction by Suction With the Duodenal Tube, J. A. M. A. **101**: 1532, 1933.
2. Iselin, H.: Zur Methodik der Pleuraempyembehandlung, Beitr. z. klin. Chir. **102**: 587, 1916.
3. Carlson, H. A., and Bowers, W. F.: Acute Empyema, Internat. Surg. Digest **18**: 131, 1934.
4. Wangensteen, O. H. : Personal communication.

PARTIAL GASTRECTOMY IN TREATMENT OF PEPTIC ULCER

F. Gregory Connell, M.D., Oshkosh, Wis.

THERE are many reasons for the unsatisfactory and conflicting results of treatment of peptic ulcer; for example, the clinical facts that ulcer may exist without symptoms and that classical symptoms may exist without ulcer may cause diagnostic errors.

The spontaneous remissions that characterize the disease and a "post hoc ergo propter hoc" philosophy may lead to a confusion in evaluating results of treatment.

Rational treatment should be based upon etiology; but peptic ulcer is probably due not to a single cause but to various influences that may disturb a proper balance between the aggressive (acid, fundal) and the defensive (alkaline, antral) factors in normal digestion.

As HCl is a constant accompaniment of peptic ulcer, it must be considered as an important element in the cause of peptic ulcer. In its absence the development of such ulcers is very infrequent. The importance of HCl is shown by the location of such ulcers, i.e., adjacent to acid-secreting areas, such as esophagus, antrum, duodenum, jejunum at gastroenterostomy stoma, ileum at Meckel's diverticulum.

Gastroenterostomy might be considered as a human experiment; the jejunal ulcer that follows in a certain percentage of cases is of great significance.

Rational treatment will seem to call for stimulation of and cooperation with the defensive mechanism, and at the same time depression of and antagonism to the aggressive factors of the stomach. The latter calls for an attempt to diminish the HCl in the gastric contents. This may be accomplished temporarily by various palliative or symptomatic methods; such as medically, by drugs, alkalies, and foods to neutralize the gastric contents; or surgically, by local excision of ulcer or resection of the distal alkaline secreting stomach. More permanent relief calls for more radical measures; such as medically, antisecretagogues, which to date are not clinically applicable; and surgically, a direct minimization of the acid secreting fundus, i.e., partial fundusectomy. To be ideally or truly radical, the treatment should interfere with the causes of the hydrochloric acid secretion, but this subject is too complex to be discussed at this time.

Partial gastrectomy, which seems to be increasingly acceptable, is interpreted differently by operators and calls for standardization.

Confusion might be reduced and the value of postoperative statistics enhanced if partial gastrectomy were divided into the following: (1) distal—pylorectomy (removal of alkaline secreting area); (2) distal and middle—partial gastrectomies (removal of alkaline and small acid se-

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creting area); and (3) distal, middle, and proximal—subtotal gastrectomies (removal of alkaline and larger acid secreting area).

Partial gastrectomy has undergone a gradual evolutionary shift from right (distal) to left (proximal) (Fig. 1).

In distal gastrectomy the ulcer, pylorus, and part of the alkaline secreting antrum are removed and gastroenterostomy is established. Acidity of the gastric contents is reduced indirectly by neutralization after regurgitation of alkaline intestinal fluids. As the ulcer is but a local manifestation of a general disease, removal of the ulcer is not always necessary. Satisfactory healing may, and does, take place without removal, after a multitude of "medical managements," gastroenterostomy, "exclusion" operations, and gastrectomy for "nonresectable" duodenal ulcer.

Fig. 1.—Shift to left in gastric resection.

Removal of, or operations upon, the pylorus interfere with the complicated, delicately balanced antropyloroduodenoneuromuscular mechanism and substitutes an artificial unanatomic and unphysiologic gastrointestinal communication. Proper preoperative treatment may often make pyloric operations unnecessary.

Removal of the antrum is based upon the gastrin theory of Edkins, i.e., antral production of a hormone that stimulates fundal acid secretion. Experimental and clinical observations indicate that such a function of the antrum is not of great importance and the eminent physiologist Ivy has suggested that gastrin and histamine are very similar, if not the same. A reason for retaining the antrum might be the preservation of beneficial secretions, such as alkali, mucus and, perhaps, an antianemic hormone.

Results after this type of surgical treatment have not been entirely satisfactory and have led to modifications and extensions such as distal and middle partial gastrectomy in which the removal of additional proximal stomach is added to the distal resection which aids in reducing gastric acidity directly by removal of a part of the stomach wall that secretes acid. But results were so frequently unsatisfactory that the removal of more and more of the fundus finally led to the deliberate performance of distal, middle, and proximal subtotal gastrectomies in which the reduction of gastric acidity is aided directly by removing more of the acid secreting stomach.

A resection to be classified, as "subtotal" should remove two-thirds or four-fifths of the stomach, the line of proximal division being at the re-entrant angle of the lesser curvature and extending to the greater curvature at right angle to the long axis of the stomach.

Remote results after distal, middle, and proximal gastrectomies seemed to be better than after pylorectomy and distal and middle partial gastrectomies; but the immediate mortality rate, in the hands of many operators, discouraged its general acceptance as a recognized treatment for peptic ulcer.

Additional improvement seems called for, but further shift to the left in partial gastrectomy is technically difficult, and complete gastrectomy, even if practicable, would be undesirable because of a resultant achlorhydria with a possible anemia or other secondary changes.

Study of the gradual evolution of this subtotal resection led to the suspicion that, perhaps, overenthusiasm had unnecessarily removed important structures (distal stomach) with resulting partial defeat of its purpose.

Certain modifications had been tried, such as the so-called exclusion operations, but with unsatisfactory secretory results, due probably to retention of the fundus and the artificial gastrointestinal communication; the local, wedge, or sleeve resection, but with unsatisfactory motor results, due probably to neuromotor disturbance at the lesser curvature; and the Billroth 1 procedure, but after extensive fundal resection it is rarely applicable; and the resultant end-to-end gastroduodenostomy is, at best, a poor substitute for nature's delicately balanced sphincter.

In 1929[1] I recommended partial gastric fundusectomy, i.e., direct minimization of acid secreting fundus but with retention of the distal stomach and its favorable secretory and motor functions. Such a procedure becomes logical and practical when it is realized that the stomach is, from physiologic, anatomic, embryologic, and evolutionary points of view, a double organ.

Removal of one part, the antrum (distal and alkaline), allowing more or less of the fundus to remain, but with resulting disturbance in secretory and motor function, is a recognized clinical procedure.

The reverse, i.e., removal of more or less of the fundus (proximal and acid), allowing the antrum and pylorus to remain with comparatively normal secretory and motor functions, seems a desirable improvement.

The technique is simple (Fig. 2). After exploration, confirmation of the diagnosis, and demonstration of a patent pylorus, the greater omentum is ligated and separated from the middle half of the greater curvature and the peritoneal cavities are walled off. With or without clamps a triangular segment of the fundus is removed, its base the middle of the greater curvature and its sides the anterior and posterior walls, extending in an upward and inward direction from the base to about one inch below the lesser curvature. The lesser curvature (the so-called Magen Strasse) is not divided. Removal of this part of the fundus is simplified by the anatomic arrangement of the vascular and neuromuscular structures of the stomach wall.

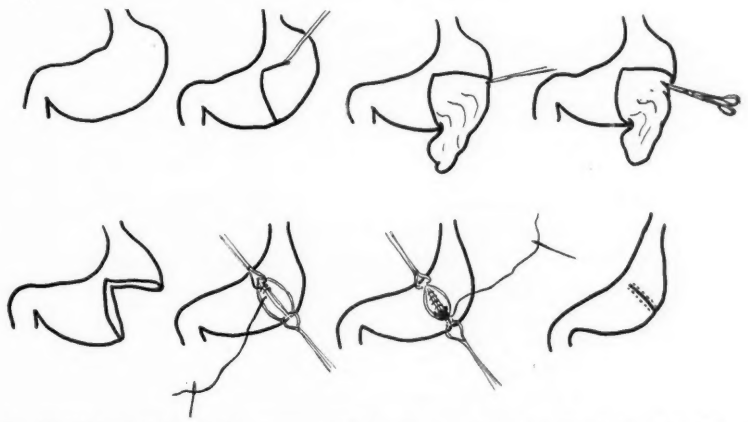

Fig. 2.—Partial gastric fundusectomy. (From Surg., Gynec. & Obst. 59: 786-788, 1934.)

Reunion of the remaining proximal and distal quarters reconstructs the stomach. By grasping the upper limit of the incision in the posterior wall and the greater curvature at the points of incision, the divided posterior stomach will be held in suitable apposition and position for hemostasis and through-and-through suturing from the upper extremity to the greater curvature. The upper extremity of the united posterior wall is released and the upper extremity of the as yet ununited anterior wall is grasped by forceps which, with the forceps remaining at greater curvature, hold the anterior wall in suitable position for over-and-over continuation of the suturing and completion of the restoration of the continuity of the stomach. Before complete closure, the gastric, pyloric, and duodenal lumen may be carefully examined. A second layer of suturing, of course, may be applied. Attaching the greater omentum to the minimized greater curvature completes the operation and the abdomen is closed without drainage. The postoperative management will be the same as may be customary after any other type of stomach operation.

After animal experimentation,[2] this procedure was carried out clinically for the first time on Dec. 14, 1931.[3]

The clinical cases have subsequently increased to fourteen. It is recognized, of course, that the number is too small and the elapse of postoperative time too short for the series to be of statistical value.

The fact that the result in the first clinical case,[3] definitely "intractable," has been entirely satisfactory for six years despite repeated psychic and physical trauma is encouraging.

Details concerning these fourteen cases follow:

Sex:	Male	12
	Female	2
Age:	20-30 yr.	1
	30-40 yr.	5
	40-50 yr.	5
	Over 50 yr.	3
Duration of symptoms:		
	None	2
	1 yr. or less	2
	1- 5 yr.	1
	5-10 yr.	
	10-20 yr.	9
Previous appendectomy		8
Previous appendectomy and gall-bladder operation		1
Previous acute perforation, gastroenterostomy		4
Previous acute perforation, simple closure		4
Previous acute hemorrhage		3
Previous degastroenterostomy		1
At operation:		
	Duodenal ulcer	9
	Jejunal ulcer	3
	Gastric ulcer	1
	Chronic gastritis	1
Partial fundusectomy, ulcer remains		7
Partial fundusectomy and excision of ulcer		5
Partial fundusectomy with pyloroplasty		1
Partial fundusectomy chronic gastritis		1
Deaths:		2
	1 on 14th postoperative day due to uremia	
	1 on 5th postoperative day due to intestinal obstruction caused by adhesive band at site of previous laparotomy for appendicitis	
Of remaining 12 cases, in 1 (No. 7) recurrence of duodenal ulcer occurred, possibly due to insufficient removal of fundus (comparable to insufficient removal of thyroid in hyperthyroidism)		

In all other cases the results have been satisfactory despite a compensatory return, within six months, to approximately normal gastric size and secretion, as was found to occur in animal experiments by Watson,[5] Seely and others,[6] Fauley and Ivy,[7] the author,[2] and others. The

advisability of supplementing the procedure with medical management and dietotherapy has been emphasized by the experimental work of Ivy.[7] In these cases the postoperative advice has been avoidance of alcohol, tobacco, and salt; frequent feedings of small amounts of no special diet.

In arriving at a diagnosis the history was found to be most important, the physical examination of comparatively little assistance, the constancy of a low blood pressure noteworthy. The laboratory findings disclosed an hyperchlorhydria. The barium meal usually revealed a deformity suggestive of ulcer.

CONCLUSIONS

1. Diminution of hydrochloric acid secretion seems to be a logical objective in the treatment of peptic ulcer.
2. Radical and palliative methods should be definitely differentiated.
3. Their combination is often advantageous.
4. Radical medical management (antisecretagogues) are not clinically applicable.
5. Radical (comparative) surgical measures (partial gastrectomy) seem to be gaining favor.
6. Partial gastrectomy may be classified as: (1) distal (pylorectomy), (2) distal and middle (partial gastrectomy), and (3) distal, middle, and proximal (subtotal gastrectomy).
7. Minimization of proximal acid fundus with preservation of the distal alkaline antrum and the pyloric mechanism is suggested as an improvement and as
8. A compromise between the conservative gastroenterostomy and the more radical, gastric resection.
9. It may, or may not, be combined with resection or destruction of the ulcer, or with plastic operations.
10. Auxiliary dietotherapy and medical management will be beneficial.
11. If the remote result is unsatisfactory, the more extensive resection may be carried out subsequently.
12. The results after such treatment in fourteen cases of peptic ulcer are reported.

REFERENCES

1. Connell, F. Gregory: Fundusectomy, Surg., Gynec. & Obst. **49**: 696-701, 1931.
2. Connell, F. Gregory: Fundusectomy, Experimental, Surg., Gynec. & Obst. **53**: 750-752, 1931.
3. Connell, F. Gregory: Resection of Fundus of Stomach for Peptic Ulcer, Ann. Surg. **96**: 200-203, 1932.
4. Connell, F. Gregory: Partial Gastric Fundusectomy in Treatment of Peptic Ulcer, Surg., Gynec. & Obst. **59**: 786-788, 1934.
5. Watson, James R.: Effect of Fundusectomy on the Acidity of the Gastric and Duodenal Content, Arch. Surg. **31**: 1-9, 1935.
6. Seely, H., and Zollinger, Robert: Fundusectomy in the Treatment of Peptic Ulcer, Surg., Gynec. & Obst., 155-161, 1935.
7. Fauley, G. B., and Ivy, A. C.: The Prevention of Post Operative Jejunal Ulcer by Diet and Fundusectomy, Surg., Gynec. & Obst. **63**: 717-723, 1936.

HEMANGIOMA AND ITS TREATMENT*

W. T. Peyton, M.D., and N. L. Leven, M.D., Minneapolis, Minn.
(From the Department of Surgery, University of Minnesota†)

THE subject of angioma should be of interest to everyone in the practice of medicine whether he be general practitioner or specialist, for when considered in all of its forms it is a common lesion involving all parts of the body. The diagnosis may be very difficult. It is frequently an unexpected finding during an operation for the removal of a benign tumor. Its treatment always will have to be varied to suit the form and location of the lesion. At the present time there is little unanimity of opinion concerning treatment.

CLASSIFICATION

Essentially there are two forms of angioma, the capillary and the cavernous, but there are many variations of each of these types. The capillary type or hemangioma simplex (Virchow) or telangiectasis, consisting of an increase in number and dilation of capillaries, appears chiefly in the form of the port-wine stain, the strawberry type, and the stellate angioma.

These lesions tend to come under the care of the dermatologist and are of interest here chiefly in so far as the port-wine and strawberry types frequently are associated with a cavernous hemangioma. The port-wine angioma consists of an increased number of dilated capillaries and venules in the skin over which the epidermis may be very thin, frequently with a velvety surface. It occurs most frequently in the face and at times is confined in its distribution to the trigeminal nerve area, stopping short at the midline. This type of angioma does not tend to spread. Such lesions in the scalp may be associated with intracranial hemangioma involving the cortical vessels.

The bright red lesion frequently with bright red excrescences known as the strawberry type is composed of capillaries with small lumina and hypertrophic endothelium. It is prone to occur at the site of embryonic fissures in the face, lip, neck, and perineum.

Cavernous angioma in its typical form resembles erectile tissue of the corpora cavernosa penis, consisting of large blood spaces or sinuses lined with endothelium. Many deviations from this typical structure occur. There is extreme variability in the amount of connective tissue between the endothelial lined spaces, and these spaces vary greatly in size. Mixed

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forms of cavernous hemangioma occur, the most common being a combination with lymphangioma. They are found most frequently in the tongue and floor of the mouth. Lipoma and neurofibroma are also seen in combination with cavernous hemangioma. It is obvious that the tumor with large blood spaces and little connective tissue stroma will be most compressible and that the one with small spaces and much connective tissue or the mixed tumor will be relatively noncompressible. This is of practical importance in selecting a form of therapy to fit the individual tumor.

CAVERNOUS HEMANGIOMA OF VARIOUS TISSUES

The clinical forms of cavernous hemangioma are as numerous as the different tissues in which they occur, and they occur in all tissues according to Kornmann,[1] except in cartilage. Several of these different forms will now be considered briefly, and their treatment will be taken up in the final section of this paper.

SKIN AND SUBCUTANEOUS TISSUES

The cavernous type occurs most frequently in the skin and subcutaneous tissue and the head is the common location.

The typical history is that they were present at birth or appeared early in life and gradually enlarged up to puberty. Eighty-three per cent of 645 cases of hemangioma were present at birth and an additional 13 per cent appeared in the first three years of life according to Fitzwilliams.[2] They appear as bluish, irregular, compressible tumors. The diagnosis, as a rule, is evident. If not discolored, the fact that they are compressible indicates the vascular nature of the tumors, and absence of a bruit or thrill demonstrates that they are not arteriovenous aneurysms.

MUCOUS MEMBRANES

Angiomata of mucous membranes occur most frequently in the oral cavity where they are often mixed with lymphangiomata. The diagnosis is made without difficulty in the visible lesions. Hemangiomata of the gastrointestinal tract are rare. In 1936 Kaijser[3] was able to find 44 cases reported and added 2 of his own. He classified them as cavernous, 22 cases; polypoid type, 10 cases; hemangioma simplex, 6 cases; and part of a generalized angiomatosis, 8 cases.

Hemangioma of the gastrointestinal tract manifests itself by repeated intraintestinal hemorrhages, usually beginning in infancy. Symptoms of intestinal obstruction may be produced by a pedunculated lesion.[4] The diagnosis is rarely made before exploratory laparotomy or autopsy[5] but can be made by proctoscopic examination if it involves the lower colon. Radiologic examination is of value if the lesion is pedunculated or contains phleboliths, but if none of these complications are present, a diagnosis can only be made tentatively on a history of persistent bleeding

over a number of years, beginning as a rule in infancy. Unsuccessful hemorrhoidectomy has been done for persistent bleeding in some of the reported cases. The one case of gastrointestinal lesion which we have encountered was a boy sixteen years of age who gave a history of never being able to play as other boys of his age because he tired so easily; but he gave no other history of symptoms which might have been related to hemangioma of the intestine until he developed measles. Following this illness he became very pale and weak. This continued for eighteen months when he suddenly collapsed, passed blood, and his hemoglobin dropped to 15 per cent, his red cell count to 900,000. All examinations, including gastrointestinal radiologic study, were negative and no cause for his bleeding was found. Abdominal exploration was made with a preoperative diagnosis of probable Meckel's diverticulum. The hemangioma was found involving the upper art of the small bowel, probably all of the jejunum, and upper part of the ileum with irregular patches of dilated veins or venous spaces in the wall of the gut.

SINUS PERICRANII

Sinus pericranii is the name most frequently applied to a cavernous hemangioma of the pericranium which communicates by one or more abnormal foramena through the skull with the dural sinuses. Although such lesions had been previously described (Percival Pott,[6] 1760, and Pellatan,[7] 1810), Stromeyer[8] in 1850 first called it sinus pericranii, and since that time it has been known as sinus pericranii of Stromeyer. Reported lesions are relatively rare. Fèvre and Modec[6] in 1936 were able to find only 64 cases, but according to Hahn,[9] 85 cases were reported prior to 1928. In origin they are congenital or traumatic according to the history of appearance,[10-12] but in both the course and findings are similar. Sinus pericranii appear as a soft, compressible, fluctuant swelling which increases in size when the venous pressure is increased, as in crying, straining, or when the patient assumes a posture with the head down.[8, 9, 12, 13] A diagnosis of meningocele is frequently made when there is no discoloration. Sinus pericranii gives few symptoms, but moderate pain and vague cerebral symptoms have been recorded.[8] The location of sinus pericranii in order of frequency is over the forehead, along the sagittal sinus, and over the occiput.[12, 14] The abnormal openings in the skull, as demonstrated by radiologic examination, are helpful in making the diagnosis. In the typical case we have observed, the diagnosis was suggested by palpation of phleboliths when the blood was drained or expressed, made by the apearance of phleboliths in radiographic film, and confirmed by aspiration of blood. In an atypical case the diagnosis was made from the history that the family physician had lanced it on the assumption that it was an abscess, and this was followed by an alarming hemorrhage.

MUSCLE

Cavernous angiomata of muscle are one of the more common forms of hemangioma. The total number of reported cases is approximately 270.[15] The diffuse type of hemangioma of muscle which is more common than the circumscribed may involve other structures, especially joints, tendons,[16] bone, nerves, or subcutaneous tissues, so that it is difficult to classify them as primary in any one of these tissues. In a personal experience with 5 cases, there was subcutaneous involvement in 2 and involvement of the knee joint in 2.

In the reported cases 95 per cent occurred in patients less than thirty years of age, and the lower extremity has been most frequently involved, 107 of 256 cases (42 per cent) being in the thigh.[17] Our experience is very much in agreement with this; all of our patients were less than thirty years of age; all were in the lower extremity, 4 in the thigh and 1 in the leg.

Tumor mass is the most constant but not always present symptom. Pain, tenderness, weakness, especially a tendency to tire, and later functional impairment, even with contraction deformities, occur. All of these symptoms were encountered in the cases we have observed. In only one was no tumor visible, but this girl did note a swelling when she first got up in the morning or when she stood still for a long time, but with muscular contraction it disappeared.

Diagnosis has been correctly made before operation in only 12 per cent of 256 reported cases.[19] Many are sufficiently compressible to make the diagnosis and, if suspected, can be confirmed by aspiration of blood. Phleboliths were demonstrated in 34 of 63 cases examined by radiologic study. In one of our cases the diagnosis was made by this finding, while in another it was not made but should have been made, since phleboliths were present in a radiograph taken before operation. For a more complete discussion of hemangioma of muscle, the reader is referred to the paper of Davis and Kitlowski.[18]

Ragnotti[20] in 1937 was able to find reports of 22 cases with primary involvement of the knee joint. In such lesions the symptoms are similar to internal derangements of the knee joint and cannot be accurately diagnosed before operation.[21-25] Two cases we have observed had knee joint capsule and possible intraarticular involvement as part of a lesion involving the lower thigh and region of the knee joint. They have been classed here as hemangioma of muscle with involvement of the knee joint.

TREATMENT

Various methods of treatment have had periods of popularity in the treatment of hemangiomata. Successful results have been reported with all methods, but failures were either so frequent or complications

so frequent and severe that many methods once advocated have been discarded. Some are no longer used because they were not fundamentally sound, and others because their use was extended to include lesions for which they were not suitable, and the method was discredited. Almost 100 years ago, injections of ferric chloride solution were introduced in the treatment of hemangioma, no distinction being made at that time between hemangioma, arteriovenous aneurysms, and even arterial aneurysms; so many disastrous results occurred that it was abandoned.

Even in very ancient times, blood vessel tumors, probably including angiomata, were treated by cauterization or excision,[26] but in the past 100 years, other methods have been introduced. Macilwain[27] in 1835 reported a case treated by the introduction of red hot needles, and, when this failed, setons armed with threads were passed. This, as well as a second case in which setons armed with threads were passed, he reported as cured. The description of the lesions indicated that they were subcutaneous cavernous angiomata of the head. Lallemand,[28] 1835, reported 4 cases, 3 treated by partial excision, repeated partial incision and suture, and incisions and suture; 1 treated by 108 punctures. The curative agent in these procedures was, as he said, scar tissue from injury and suppuration.

Pravez[29] prior to 1845 seems to have introduced or popularized the injection of ferric chloride for coagulation of blood in aneurysms and hemangiomata, since it is frequently referred to as the treatment of Pravez in reports published about this time by Jobert,[30] Pravez,[29] Valette,[31] Lenoir,[32] Alford,[33] Soule,[34] Giraldes,[35] and Follin.[37] Lusana[38] in 1845, using acetate of peroxide of iron instead of ferric chloride, reported the cure of a case of hemangioma. That the injection of ferric chloride was at times successful, even in arterial aneurysm, is indicated by the case reported by Valette.[31]

Vaccination was introduced about this same time for the destruction of hemangioma by its suppuration and scar formation.[26] It was revived by Friedjung[39] in 1933 but is obviously useful only in small lesions.

Insertion of heated needles was still used in 1850 as indicated by a report of two cases successfully treated in this manner.[40]

Injection of hot water was introduced by Wyeth[41] in 1902 and since then has been extensively used by others.[42] Wyeth reported that in addition to capillary angioma and venous angioma (cavernous) he also had successfully treated cirsoid aneurysms by the injection of hot water.

Alcohol injections for the cure of angiomata were reported by Gallant[43] in 1903; 67 cases were included in his report, and in general he considered the results satisfactory. This method is now being used at the Minneapolis General Hospital in the Department of Dermatology under the direction of S. E. Sweitzer with satisfactory results in properly selected lesions.

Galvanocautery introduced in 1854 by Middeldorff was soon used in the treatment of hemangioma and more recently the high frequency current has been used. Figi[44] in 1936 considered it the method of choice in treatment of cavernous hemangiomata of the face. We have tried it but with results so inferior to those following injection of sclerosing solutions that it has been discontinued. Carbon dioxide snow introduced by Pusey[45] in 1907 is still used extensively by dermatologists in treating superficial lesions.[46, 47]

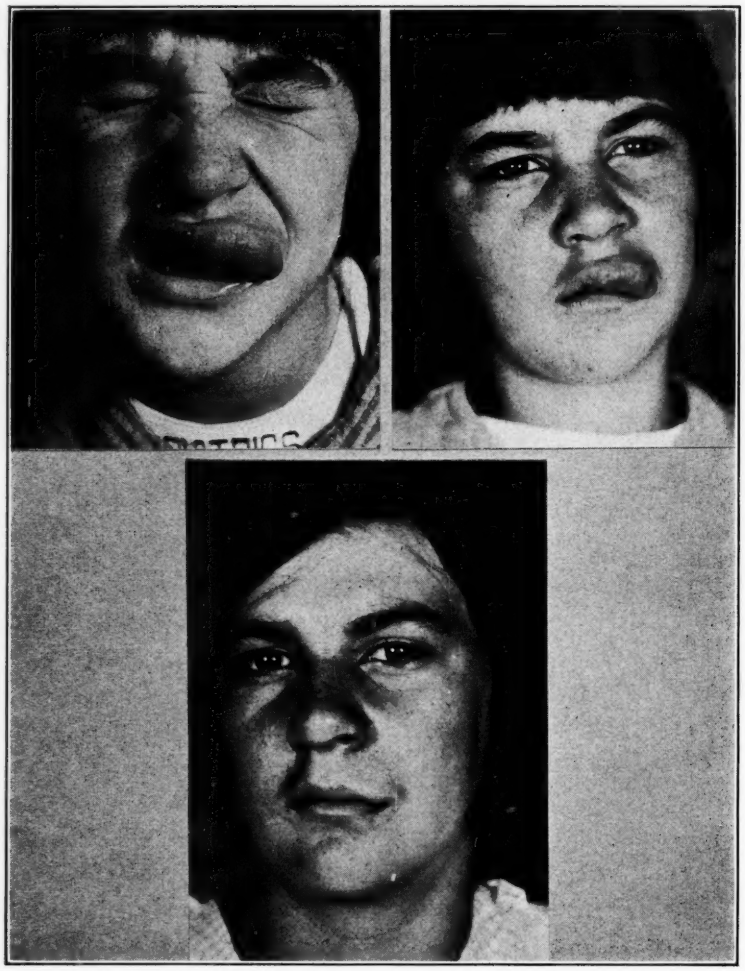
Irradiation introduced in comparatively recent times is still extensively used and with success in properly selected cases.[48] It is most effective in hemangiomata of children. It does not destroy the port-wine stain lesion in safe dosage. Cavernous hemangiomata in children at the University of Minnesota Hospitals are, for the most part, referred to the Department of Roentgen and Radium Therapy, but lesions in children unsuitable for the application of x-ray or radium, for example a lesion completely occluding the external auditory canal or in the tongue, as well as other lesions in children which failed to respond to radiation therapy, have been successfully treated by sodium morrhuate injections.

Excision has been practiced all through the period in which these other methods have been introduced and is still the method of choice by some surgeons (Lloyd,[49] 1929; Levi,[50] 1930; Kaplan,[51] 1932; Davis and Wilgis,[52] 1934; and Petrov,[53] 1934). Others consider surgical treatment only as an adjunct to other forms of treatment. In the circumscribed or encapsulated form surgical removal certainly is a satisfactory procedure and the only objection is the disfigurement of the resulting scar. It should be employed when indicated.

Babcock[54] in 1917 injected quinine hydrochloride and urethane in the treatment of hemangiomata, and Dowling[55] in 1929 reported 5 cases treated by quinine dihydrochloride and urethane. Injection of sclerosing solutions for the cure of hemangiomata frequently is referred to as Dowling's treatment. Other reports concerning the use of sclerosing solutions in the treatment of hemangioma have been made by Porritt,[56] Carabba,[57] McEvedy,[58] Berthwistle,[59] Sesary,[60] Andrews and Kelly,[61] and Demel.[62] Still others in recent reports merely mention injection as a method of treatment but do not particularly recommend it.[44, 63, 65-69] That the injection of sclerosing solutions is not a fully accepted type of therapy is suggested by the appearance in 1936 of a monograph on oral surgery that does not even mention this method but recommends only Wyeth's injection of boiling water;[70] the *Journal of the American Medical Association*, Nov. 14, 1936,[71] in answer to a question concerning the treatment of cavernous hemangioma does not mention injection treatment; and Figi[44] in 1936 recommends electrocoagulation as the preferable procedure for treatment of these lesions when located on the face.

REVIEW OF CASES TREATED AT UNIVERSITY OF MINNESOTA HOSPITALS

The records of 132 cases of hemangioma treated by Roentgen ray and radium at the University of Minnesota Hospitals since April, 1929, have been reviewed. One hundred and nine were treated with superficial Roentgen therapy with excellent results in 22; good, in 33; and poor to fair, in 40; no follow-up record was made in 14. Therefore, approxi-



A. *B.*

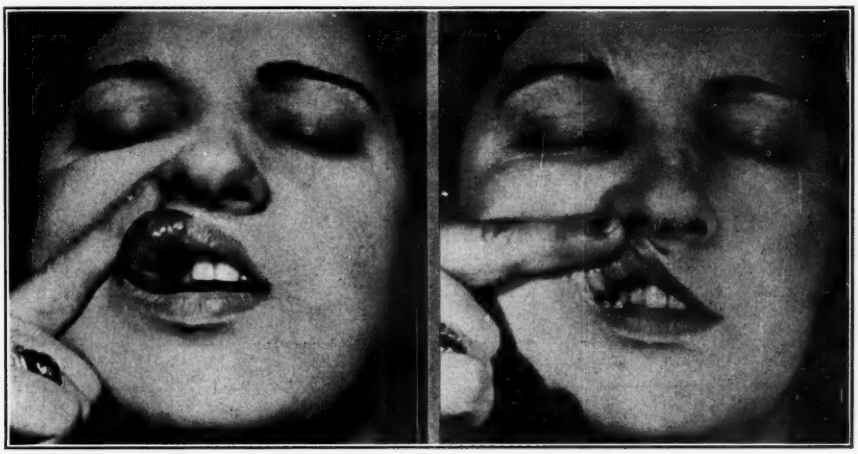
C.

Fig. 1.—*A*, Cavernous hemangioma, girl nine years old; *B*, after x-ray therapy a fibrous tissue mass remains; *C*, after removal of fibrous tissue mass by plastic operation with incision on the intraoral side of lip.

mately 51 per cent of the traced patients obtained excellent or good results. Dividing the material into patients less than fifteen years of age and over fifteen years of age, we have treated 75 cases in which the age was recorded as less than fifteen years. Sixty-eight of these were traced and 40, or 53 per cent, obtained an excellent or good result. Twenty-

three patients were treated with high voltage therapy and of these the results were excellent in 5; good, in 6; poor, in 9; and no follow-up record was made in 3. Therefore, approximately 48 per cent of the traced patients treated with high voltage x-ray therapy received excellent or good results. Divided into patients over and under fifteen years of age, the results on those treated with high voltage x-ray therapy were better in the young group.

The injection of sclerosing solution was first used in the treatment of hemangioma at the University of Minnesota Hospitals in April, 1929, and the records of 33 cases treated by this method have been reviewed. In 15 the results were excellent; in 8, good; in 5, not good or resulted in failure; and 5 were not followed or are still under treatment. Therefore, approximately 82 per cent of the followed patients obtained excellent or good results by this treatment.



A. *B.*

Fig. 2.—*A*, Cavernous hemangioma in right upper lip and cheek; *B*, hemangioma obliterated by injections of sodium morrhuate.

TREATMENT OF CUTANEOUS AND SUBCUTANEOUS HEMANGIOMA

The port-wine and strawberry type are referred to the Department of Dermatology and are not considered here except in so far as they are included in the cases given x-ray therapy.

The cavernous hemangioma of subcutaneous tissues must be compressible to be successfully treated by the injection of sclerosing solutions, all of which injections, when successful, have been made into the blood spaces. In adults where the spaces have become large and the skin has become thin over these spaces, the results have been most satisfactory. A few subcutaneous encapsulated lesions were excised as a primary procedure and some nonencapsulated subcutaneous lesions were excised following x-ray therapy or injection.

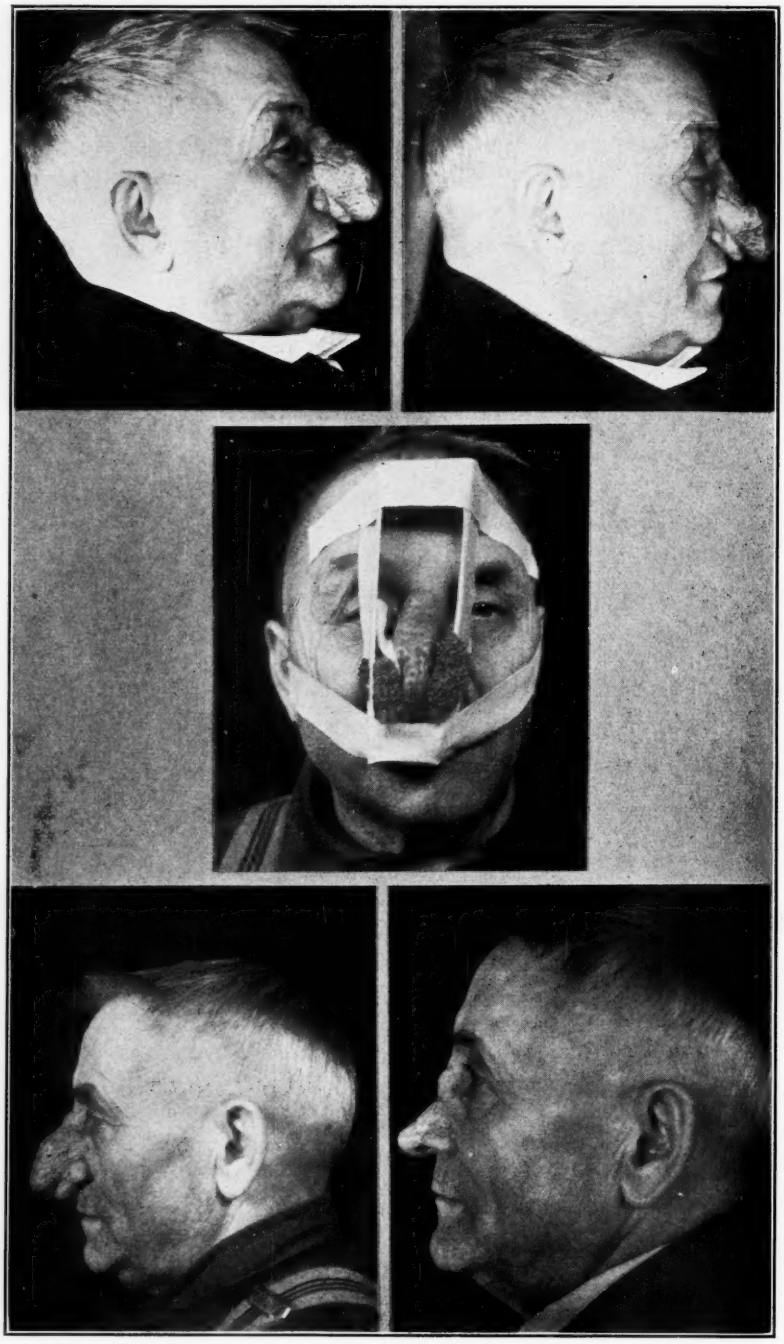


Fig. 3.—*A*, Arteriovenous aneurysm and secondary rhinophima; *B*, the blood spaces emptied by compression; *C*, compression is applied with rubber sponge and tongue blades following each injection; *D*, the blood spaces have been obliterated by injections of sodium morrhuate; *E*, the rhinophima has been removed; bleeding was not inordinate.

TREATMENT OF HEMANGIOMA OF MUCOUS MEMBRANES

Two cases of hemangioma of the tongue, one involving also the floor of the mouth and cheek, were injected with excellent results. One case of intestinal hemangioma was encountered at abdominal exploration and later given high voltage x-ray therapy. The patient was reported some eight months later to have had no more gross hemorrhages. It may be advisable when the boy—now sixteen years of age—is older to resect some or all of the involved portion of small bowel, a procedure reported by Kaijser[3] as successful in the cure of a hemangioma of the sigmoid. The only report of injection of sclerosing solution for the cure of hemangioma of the bowel found in the literature is that of Bancroft,[72] who successfully injected a hemangioma involving the superior hemorrhoidal vein. He ligated the vein and injected on the peripheral side of the ligature. Eleven months later on reopening the abdomen, all evidence of the lesion was gone. In spite of this successful result it would seem that injection of sclerosing solutions for cure of gastrointestinal hemangioma would probably have a very limited field of application. In our experience few angiomata, unless very small, have disappeared following one injection.

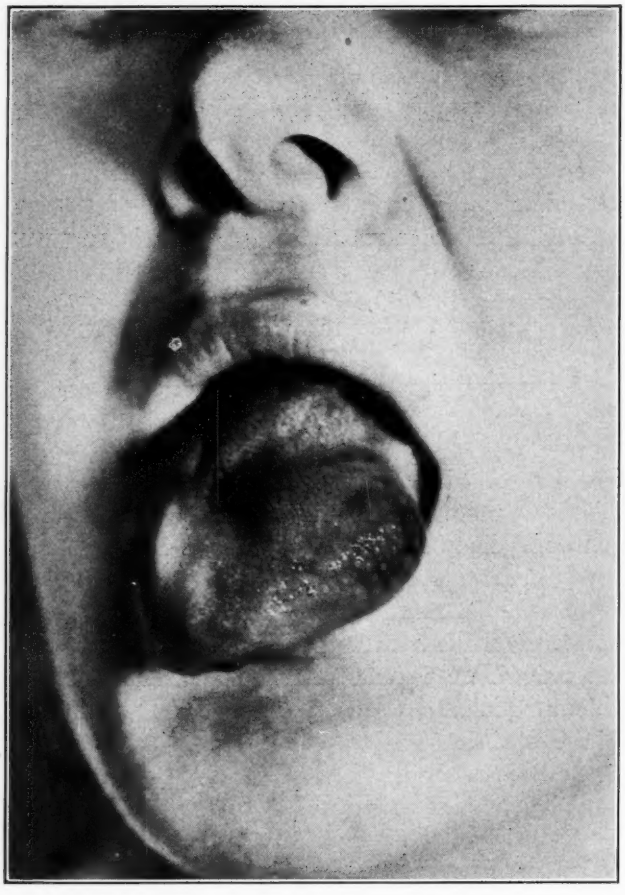
TREATMENT OF SINUS PERICRANII

A typical case of what is now known as sinus pericranii was treated by Moreau and reported by Pelletan[7] in 1810. Moreau, without stating what he thought of the nature of the lesion or what he intended to do, cut the tumor through the middle; it bled freely but the two portions of the lesion did not decrease in size. It was packed with lint but continued to bleed. Antimony paste was applied and finally the bleeding was controlled, but the child died the fourth day after operation of symptoms typical of meningitis. Postmortem examination demonstrated large communications with the superior longitudinal sinus. Mastin[73] in 1886 found in the literature 12 cases which had remained stationary when followed from five to twelve years, and 7 which had had operative procedures with 4 deaths—2 from hemorrhage and 2 from suppuration. It was his conclusion that surgical interference was not indicated, but, if deemed expedient or demanded, operation should be limited to ligation of pedicle, ligation of the sinus, or electropuncture.[74]

The first successful operation for sinus pericranii, according to Cohn,[12] was by Franke[75] in 1902. Since then many have been successfully operated upon (Mueller,[76] Fevre,[77] Hahn,[9] Sudhoff,[78] and Hedry[79]). Fevre and Modec[6] find that 19 cases have been treated by ablation of the sinus pericranii and blocking of the communications with no deaths, and in 15 cases in which the results are given, 13 were successful.

We have treated one case of sinus pericranii by the injection of sodium morrhuate with an excellent result, not even a scar remaining. No reported cases treated by this method have been found. The objection

to this method might be raised, as it is raised by Fevre and Modec[6] against electrocoagulation, that thrombosis may extend into and obliterate the sinus. When the lesion overlies and communicates with one of the more important sinuses as the posterior part of the superior longitudinal, there may possibly be danger of such a complication; but the same objection has been raised against injection of sclerosing solutions into angiomata about the face and seems to have proved entirely groundless.



A.

Fig. 4.—*A*, Cavernous hemangioma of left side of tongue; *B*, hemangioma obliterated with injections of sodium morrhuate.

After an experience of several hundred individual injections of sodium morrhuate, we have never seen a thrombosis extend beyond the lesion itself. The structure of a hemangioma is so different from that of a varicose vein that the resulting thrombosis following injection in hemangioma is much more localized. It is our opinion that this is due to small communications between large venous spaces. Wax reconstructions are now being made to verify this.

One atypical case of sinus pericranii in an adult we have treated with x-ray. This lesion was atypical in that it had a very small tumor mass outside the skull over the forehead and communicated with the cranial cavity by an opening approximately 1½ c.c. in diameter, giving the impression that it was a part of a larger intracranial angioma. It did respond to x-ray therapy so that all visible and palpable evidence of the tumor and defect in the skull disappeared, but he has had return of headache and other symptoms of mild increased intracranial pressure, probably due to the intracranial portion of the lesion.

Fig. 4-*B*.—See opposite page for legend.

TREATMENT OF HEMANGIOMA OF MUSCLE

No favorable reports of the treatment of hemangioma of muscle by injection of sclerosing solutions have been found in the literature. Stewart and Bettin,[21] 1924, state that severe local inflammation, gangrene, multiple emboli, and death have followed the coagulants that were then in use, such as boiling water or astringent chemicals of which they mention ferric chloride. Jenkins and Delaney,[80] 1932, mention excision as the only proper type of treatment for hemangioma of muscle. McDermott,[19] 1935, says ligation of vessels, x-rays, radium, and injection of sclerosing solutions have all been found inefficacious in the treatment of hemangioma of muscle. Kidner,[15] 1936, states that coagulation by injection of chemicals has been tried with success in the small ones, but in large ones

the difficulty of reaching all parts of the tumor and the danger of necrosis and poisoning have led to its abandonment, and radiation, he says, has not yet proved of value. He recommends surgical removal in more than one stage if necessary.

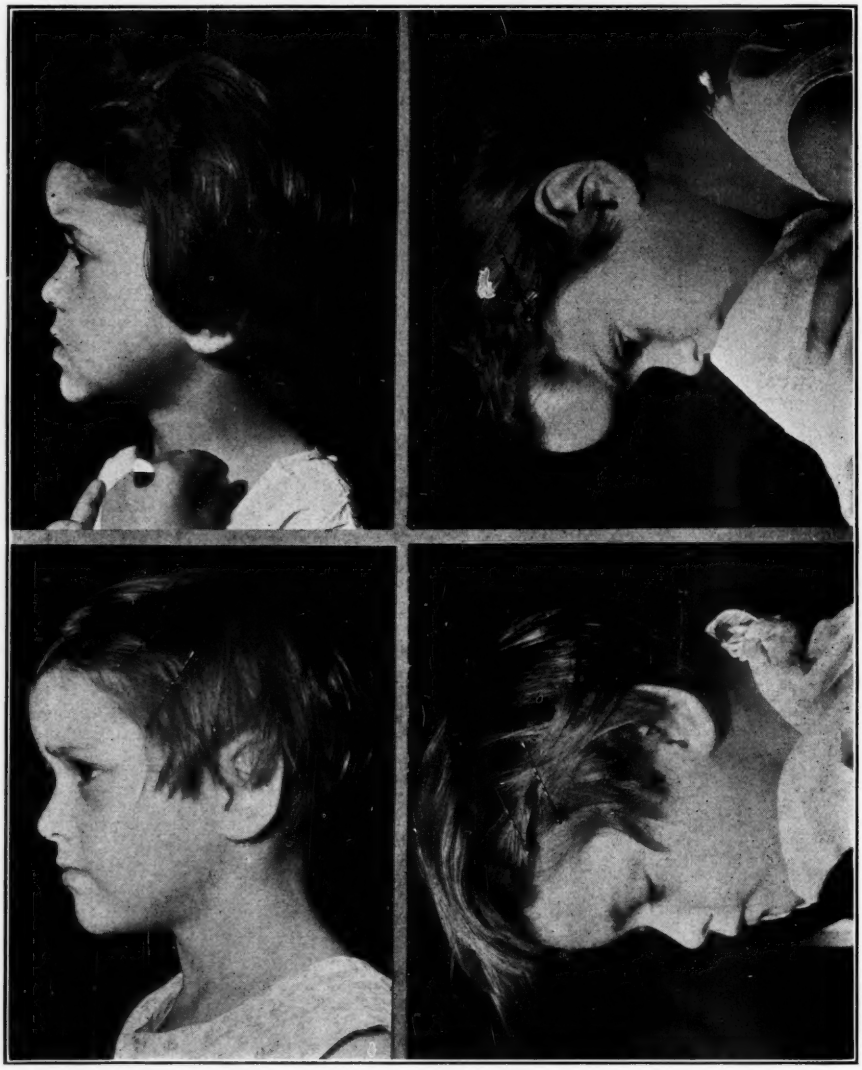


Fig. 5.—*A*, Sinus pericranii partially emptied in upright position; *B*, sinus pericranii filled in dependent position; *C*, sinus pericranii obliterated by injections of sodium morrhuate; *D*, sinus pericranii obliterated by injections of sodium morrhuate.

Our experience in the treatment of hemangioma of muscle is not large and the results of injection of sodium morrhuate have not been excellent, but on the whole they have been quite satisfactory. The first case of muscle hemangioma observed was in the gastrocnemius and soleus

muscles. A biopsy was attempted and resulted in an alarming hemorrhage; x-ray therapy gave no improvement; injection was not considered at that time and amputation was performed. Since then 4 other patients have been seen and treated by sodium morrhuate injections, 2 with definite muscle hemangioma and 2 not so definitely involving muscle but with involvement of the lower thigh, knee joint capsule, and probably knee joint. Because these latter 2 patients were not operated upon by us, we cannot be certain that the muscles were involved, but it is probable that they were in part, and these lesions have been included under hemangioma of muscle. The 2 with definite muscle involvement are still under treatment but their subjective symptoms have been much improved. In only 1 was a tumor mass present and this has been

A. *B.*

Fig. 6.—*A*, Perigeniculate hemangioma with 20 per cent limitation of extension in left knee; *B*, partial obliteration by injection of sodium morrhuate solution; full extension of left knee.

partially reduced. The 2 with perigeniculate lesions had compressible tumors which have been obliterated and practically all symptoms relieved. Full function in the knee joint has been restored. We have no experience with localized lesions in muscle, but it would seem that excision would be the method of choice in such a lesion.

TECHNIQUE FOR INJECTION OF SCLEROSING SOLUTION

The technique of injection is very simple. Sodium morrhuate in 7 per cent solution has been used and the injection is made into a blood space. To get the point of the needle into these vascular spaces, unless they are very superficial, it is necessary to plunge a short beveled needle well into, or through, the tumor and withdraw slowly with suction in the

needle until blood is withdrawn. The space is then emptied as much as possible by elevation or compression or both and solution is injected in amounts varying from a few drops to 2½ c.c. Manual compression is maintained until a rubber sponge has been bandaged over the area to maintain compression.

In subcutaneous angioma the patient experiences a stinging or burning painful sensation in, and for some distance around, the site of injection. This burning sensation subsides in a few minutes after the injection is made, but pain continues as a rule for an hour or more. It is our impression that this residual pain is less severe if moderate compression is maintained than if it is not compressed. In some lesions, especially those of the face, an overfilling seems to follow immediately after injection and in our early experience some of these were alarming. One of the first cases injected was in an extensive lesion of the lower lip and after injection of 2 c.c. of sodium morrhuate and without subsequent pressure the entire lower lip increased to two or three times its former size, became discolored and edematous so that we feared slough of the entire lip, but it eventually subsided with no slough and cure of the angioma. We have now come to realize that these reactions are not dangerous, no slough having occurred in any case in which the solution was injected into a blood space, but they are avoided by injection of smaller quantities of solution and by the application of pressure.

REFERENCES

1. Kornmann: Die Hämangiome, Odessa, 1913; ref. absts., Zentralbl. f. Chir. **40**: 1427, 1913.
2. Fitswilliams, D.: The Etiology of Naevi: Nerve Influence of Their Causation, Brit. M. J. **2**: 489, 1911.
3. Kaijser, R.: Über Hämangiome des Tractus Gastrointestinalis, Arch. f. klin. Chir. **187**: 351, 1936.
4. Ackerman, L.: Cavernous Hemangiomata of the Small and Large Bowel, Am. J. Cancer **30**: 753, 1937.
5. Brown, A.: Vascular Tumors of the Intestine, Surg., Gynec. & Obst. **39**: 191, 1924.
6. Fèvre, M., and Modec, L.: Sinus Pericranii et Tumeurs Vasculaires Extra-craniennes Communiquant avec la Circulation Intra-cranienne, J. Chir. **47**: 561, 1936.
7. Pelletan, P. J.: Tumeur Varigueuse située en Haut de la Région Frontale, et Communiquant dans le crâne; Cautérisation; mort du malade, Clin. Chir. **2**: 70, 1810.
8. Stromeyer: Ueber Sinus Pericranii, Deutsche Klin. **11**: 160, 1850.
9. Hahn, E.: Sinus Pericranii (Reducible Blood Tumor of the Cranium), Arch. Surg. **16**: 31, 1928.
10. Mastin, W.: Venous Blood Tumors of the Cranium, J. A. M. A. **7**: 309, 1886.
11. Lannalongue: Note Sur les Tumeurs Sanguines du crâne Communiquant avec le Sinus Longitudinal Supérieur, Rev. de Chir. **6**: 918, 1886.
12. Cohn, I.: Sinus Pericranii (Stromeyer), Surg., Gynec. Obst. **42**: 614, 1926.
13. Wislicenus, U.: Über Sinus Pericranii, Inaug. Diss., Zurich, 1869.
14. Demme, H.: Ueber Extracrainelle mit den Sinus durae Matris Communicirende Blutcysten, Virchows Arch. f. Path. Anat. **23**: 48, 1862.
15. Kidner, F.: Diffuse Cavernous Angioma of the Leg, Surg., Gynec. & Obst. **63**: 647, 1936.
16. Harkins, H. N.: Hemangioma of a Tendon or Tendon Sheath, Arch. Surg. **34**: 12, 1937.

17. Mailer, R.: Traumatic Hemangiomatous Tumors of Skeletal Muscle, Brit. J. Surg. **23**: 245, 1935.
18. Davis, J., and Kitlowski, E.: Primary Intramuscular Hemangiomas, Arch. Surg. **20**: 39, 1930.
19. MacDemott, E.: Two Cases of Hemangioma of Voluntary Muscle, Brit. J. Surg. **23**: 252, 1935.
20. Ragnotti, E.: L'angioma della Capsula Articolare del Ginocchi, Arch. ital. di Chir. **46**: 189, 1937.
21. Stewart, S., and Bettin, M.: The Motor Significance of Haemangioma, Surg., Gynec. & Obst. **39**: 307, 1924.
22. Haas, A.: Über Gefässtumoren der Kniegelenkkapsel, Deutsche Ztschr. f. Chir. **173**: 130, 1922.
23. Osgood, R.: Tuberculosis of the Knee Joint. Angioma of the Knee Joint, S. Clin. North Amer. **1**: 665, 1921.
24. O'Ferrall, J.: Hemangiomas of the Knee Joint, J. A. M. A. **85**: 505, 1925.
25. Mizuno, I.: Über einen Fall von Kneiegelenkkapselangiom, Zentralbl. f. Chir. **64**: 353, 1937.
26. Sonntag, E.: Die Hämangiome und ihre Behandlung, Erg. d. Chir. u. Orthopädie **8**: 1, 1914.
27. Macilwain, G.: Deep Seated Naevus. Treated by the Introduction of Setons, Medico Chirurg. Trans. of London **18**: 189, 1833.
28. Lallemand: Observations relatives à divers Procédée Operatorés employé contre les Tumeurs Erectiles, Arch. gen. de Méd. **8**: 5, 1835.
29. Pravez, J.: Sur un Nouveau Moyen d'operer la Coagulation du Sang dans les artères, applicable à la guerison des anéurismes, Compt. rend. Acad. de Sc. **36**: 88, 1853.
30. Jobert: Nouveau cas d'Anéurysme de l'artere brachiale gueri par l'injection du perchlorure de fer, Bull. Gen. de thérap. **46**: 443, 1845.
31. Valette: Examen Anatomique d'une Piece Provenant d'un Aneurysme traite avec succes par l'injection de perchlorure de fer, Bull. Gen. de thérap. **46**: 36, 1854.
32. Lenoir: Traitment des Aneurysmes par l'injection du perchlorure de fer, Bull. Soc. de Chir. de Paris **4**: 170, 1853-54.
33. Alford: Perchlorure de Fer. Nouvelles Expériences sur les Injections de ce sel dans les Arteres, Bull. Gen. de thérap. **46**: 379, 1854.
34. Soule: Bull. de Soc. de Chir. de Paris **4**: 172, 1853-54.
35. Giraldes: Bull. de Soc. de Chir. de Paris **4**: 340, 1853-54.
36. Giraldes: Tumeur Veineuse du cou. Injection de six gouttes de perchlorure de fer Guerison, Bull. Gen. de thérap. **46**: 88, 1854.
37. Follin: Du Traitement des Varices par les Injections de perchlorure de fer, Bull. Soc. de chir. de Paris **4**: 170, 1853-54.
38. Lusana: Cure Radicale d'un Anéurisme par Injection d'acetate de sesquioxyde de fer, Compt. rend. de l'Acad. des Sc. **38**: 303, 1854; abst. Bull. Gen. de thérap. **46**: 188, 1854.
39. Friedjung: Zur Therapie des Naevus Vasculosus beim Kinde, Wien Klin. Wchnschr. **46**: 1520, 1933.
40. Schmidt, J., and Fitch, W.: Two Cases of Morbid Erectile Tissue Treated Successfully by Needles, New York M. J. **4**: 66, 1850.
41. Wyeth, J.: The Treatment of Vascular Neoplasms by the Injection of Water at a High Temperature, New York M. J. **76**: 969, 1902.
42. Reder, F.: The Treatment of Cavernous and Plexiform Angiomata by the Injection of Boiling Water (Wyeth Method), St. Louis, 1918, The C. V. Mosby Co.; also in Surg., Gynec. & Obst. **21**: 61, 1915.
43. Gallant, A.: Alcohol Injections in the Cure of Angiomata, New York M. J. **78**: 966, 1903.
44. Figi, F.: Treatment of Angioma of the Face, Arch. Otolaryng. **24**: 271, 1936.
45. Pusey, W.: The Use of Carbon Dioxide Snow in the Treatment of Naevi and Other Lesions of the Skin, J. A. M. A. **49**: 1354, 1907.
46. Fabry, J.: Zur Klinik und Behandlung der Hämangiome, Dermat. Wchnschr. **89**: 1906, 1929.
47. Semon, H.: Treatment With Carbon Dioxide Snow Pencil, Lancet **226**: 1167, 1934.
48. Marin, A.: La Radium Puncture des Angiomes Tubereux, Union médicale de Canada **63**: 171, 1934.
49. Lloyd, E.: Correspondence, Lancet **217**: 1331, 1929.
50. Levi: Discussion of paper of Porritt.[56]

51. Kaplan, I.: The Treatment of Hemangiomas, Arch. Pediat. **49**: 548, 1932.
52. Davis, J., and Wilgis, H.: The Treatment of Hemangioma by Excision, South. M. J. **25**: 283, 1934.
53. Petrov, N.: Zur Behandlung der Kavernösen Hämangiome der Zunze, Zentralbl. f. Chir. **61**: 2194, 1934.
54. Babcock, W.: The Destruction of Angiomas and Other New Growths by the Injection of Quinine and Urea Hydrochloride, New York M. J. **105**: 385, 1917.
55. Dowling, G.: A Simple Method of Treating Cavernous Naeve, Lancet **217**: 1251, 1929.
56. Porritt, A.: The Injection Treatment of Hydrocele, Varicocele, Bursae and Naevi, Proc. Roy. Soc. Med. **24**: 971, 1931.
57. Carabba, V.: Sclerosing Injections in Surgery, Ann. Surg. **99**: 668, 1934.
58. McEvedy, P. G.: Recent Advances in Injection Treatment, Clin. J. **61**: 283, 1932.
59. Berthwistle, A.: Cavernous Naevi, Lancet **226**: 22, 1934.
60. Sesary, A.: Traitement des Angiomes Tuberaux par les Injectiones Sclerosantes de Salicylate de Soude, Presse méd. **41**: 259, 1933.
61. Andrews, G., and Kelly, R.: Treatment of Vascular Naevi by Injection of Sclerosing Solutions, Arch. of Dermat. & Syph. **26**: 92, 1932.
62. Demel, R.: Ein Vorschlag zur Blutsparenden operativen Behandlung der Hämangiome, Deutsche Ztschr. f. Chir. **241**: 166, 1933.
63. Schmidt, O.: Beitrag zur Klinik und Chirurgischen Behandlung des Angioma Arterale Racemosum besonders des Kopfes, Beitr. z. klin. Chir. **118**: 178, 1920.
64. Petterson, A., and Romanus, R.: Ein Fall von Haemangioma Cavernosum Congenitum mit Tödlichen Ausgang, Acta Pediat. **14**: 417, 1933.
65. Couch, J.: Hemangioma With Fracture Through the Invaded Bone, Canad. M. A. J. **33**: 416, 1935.
66. Traub, E. E.: Discussion. Transactions of New York Dermatological Society, Arch. Dermat. & Syph. **24**: 709, 1931.
67. MacCallum, D.: Treatment of Hemangiomas, Am. J. Surg. **29**: 32, 1935.
68. Light, S.: Injection Treatment of Cavernous Hemangiomas, Arch. Dermat. & Syph. **24**: 992, 1931.
69. Annotations: Treatment of Naevi, Lancet **226**: 36, 1934.
70. Blair, V., and Ivy, R.: Essentials of Oral Surgery, St. Louis, 1936, The C. V. Mosby Co.
71. Queries and Minor Notes, J. A. M. A. **107**: 1660, 1936.
72. Bancroft, F. W.: Hemangioma of Sigmoid and Colon, Tr. Am. S. A. **49**: 458, 1931.
73. Mastin, W.: Venous Blood Tumors of the Cranium, J. A. M. A. **7**: 368, 1886.
74. Mastin, W.: Venous Blood Tumours of the Vault of the Cranium Communicating With the Intracranial Venous Circulation, Especially Through the Medium of the Superior Longitudinal Sinus, Ann. Surg. **1**: 525, 1885.
75. Franke, F.: Über die Blutcysten am Schädel und ihre Behandlung, Centralbl. f. Chir. (Beilage) **19**: 26, 1902.
76. Mueller, A.: Ueber Sinus Pericranii, Berl. klin. Wchnschr. **49**: 1372, 1912.
77. Fèvre, M.: Un cas de sinus Pericranii, Bull. Soc. de Pediat. de Paris **34**: 190, 1936.
78. Sudhoff, W.: Über ein neue einfache Operationsmethode des Sinus Pericranii (Wachsplombe), Deutsche Ztschr. f. Chir. **186**: 98, 1924.
79. Hedry, N.: Ein neues Verfahren bei der operativen Behandlung des Angioma Racemosum Capitis, Zentralbl. f. Chir. **64**: 22, 1937.
80. Jenkins, H., and Delaney, P.: Benign Angiomatous Tumors of Skeletal Muscles, Surg., Gynec. & Obst. **55**: 464, 1932.

THE OPERATIVE TREATMENT OF HYPOSPADIAS*

With a Report of 13 Cases

C. D. Creevy, M.D., Minneapolis, Minn.

(From the Division of Urology in the Department of Surgery of the Medical School, Minneapolis)

INTRODUCTION

HYPOSPADIAS is a congenital absence of the floor of the urethra beginning at its distal end and extending for a variable distance toward the perineum. The roof of the urethra is present, but that portion of the corpus spongiosum belonging to it is fibrous and contracted and causes a bowing of the penis with its concavity on the ventral surface (congenital chordee). The condition results from failure of fusion of the urethral folds in the embryo, the cause of which is not known. The frequent occurrence of hypospadias in pseudohermaphroditism points to some dysfunction of the endocrine glands, perhaps of the anterior lobe of the pituitary, as a cause of the former, but this is pure speculation.

The four recognized clinical types include the glandular, the penile, the penoscrotal, and the perineal. The latter is the type seen in pseudohermaphroditism.

The glandular type constitutes no disability and requires no treatment unless the patient develops a morbid dissatisfaction with its appearance. The operations designed for its correction will not be discussed.

In the other three types, however, the bowing of the penis constitutes a serious obstacle to normal sexual activity and, together with the misplaced urinary meatus, causes functional sterility. Moreover, as the patient grows older he becomes conscious of his deformity, while his inability to micturate in the normal masculine manner attracts unwelcome attention from his companions. Therefore the need for surgical repair of these deformities has long been evident.

OPERATIVE TREATMENT

A satisfactory operation must attain two distinct ends; namely, straightening the penis and advancing the urinary meatus to or near the normal position. This invariably requires at least two operations, because the straightening leaves, until completely healed, a poor field for construction of the urethra.

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It is usually recommended that the penis be straightened as early in childhood as possible for fear that the corpora cavernosa may become permanently deformed if the operation be deferred until adult life; in my experience it has been perfectly feasible as late as twenty-five years.

It is most convenient to defer the urethroplasty until the penis is large enough to make plenty of skin available, particularly since it is not perfectly certain that the caliber of the artificial urethra will increase with growth of the individual if the operation is done early in childhood.

DIVERSION OF THE URINE

Whatever operation is used, it is necessary to divert the urine from the operative field during healing. This may be accomplished either by cystostomy or by perineal urethrostomy. Since it is helpful to attach the glans penis by means of sutures and a rubber band to the skin of the lower abdomen during healing, as suggested by Young, perineal urethrostomy is more convenient, but it carries with it some danger of fistula formation. This can be minimized by making the urethrostomy far back in the perineum close to the anterior margin of the anus in order to have as much tissue as possible around the fistula when the tube is removed.

Cecil and others advise the use of an occlusive nonabsorbable ligature about the urethra distal to the urethrostomy (or cystostomy) during healing to prevent contamination of the wound if the tube should become clogged.

STRAIGHTENING THE PENIS

The first attempt to straighten the penis was probably that of Mettauer, of Virginia, in 1842. He divided the fibrous rudiment of the corpus spongiosum subcutaneously. Duplay, in 1874, developed the still current method (Fig. 1) of transverse incision upon the ventral surface of the penis just anterior to the hypospadiac meatus, with excision of the fibrous distal segment of the corpus spongiosum, and longitudinal closure of the fascia and skin. This must at times be supplemented by a longitudinal incision through the ventral skin and Buck's fascia just behind the glans, with transverse closure to equalize the lengths of the dorsal and ventral surfaces.

Occasionally the corpus spongiosum of the intact portion of the urethra is also fibrous and shortened. In this event excision of the distal incomplete segment will not straighten the penis, a situation which can be recognized during the operation. It is corrected by dissecting the urethra and its meatus entirely free from the corpora cavernosa and skin until the penis can be pulled out straight by gentle traction. The meatus is then allowed to retract as far beneath the skin as it will, and a new opening is made for it in the penoscrotal angle. I have not seen this fact emphasized in any discussion of hypospadias, but I believe that its

recognition and correction are of fundamental importance in straightening the penis when the bowing is severe.

McIndoe states that it is impossible in some instances to straighten the penis completely by Duplay's method, in which case he recommends the more complicated two-stage method of Edmunds (Fig. 2). This operation has not yet been required in my experience, perhaps because the urethra has been separated from the corpora and allowed to retract in the more severe cases.

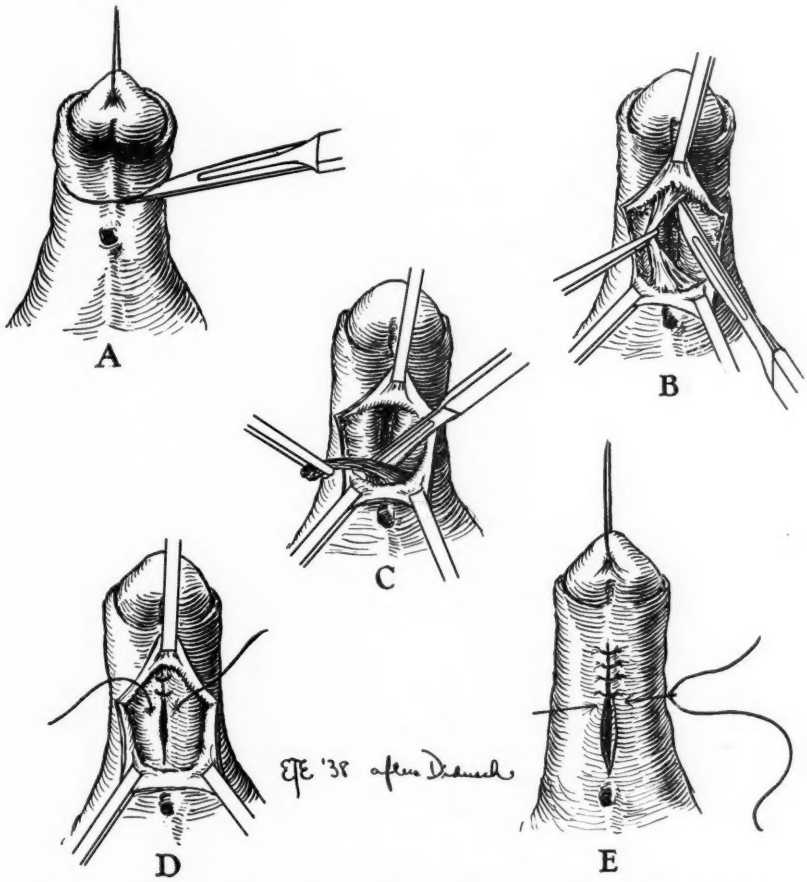


Fig. 1.—Duplay's method of straightening the penis modified from Cecil.[4]

A minimum of six months should elapse before construction of the urethra is begun, for fear that scar tissue will form, contract, and reproduce the curvature.

CONSTRUCTION OF THE NEW URETHRA

That this is not so simple is indicated by the number of methods devised for its accomplishment. The earliest attempts involved the free transplantation of tissues either from an animal, from another person,

or from the patient himself. Tissues used included the saphenous or some other vein (Tuffier, 1899), the ureter (Schmieden, 1909), the appendix (Weitz, 1915), and tubes of skin (Nové-Josserand, 1897) or of vaginal mucosa (Legueu, 1918). While temporary success was obtained at times by some of these methods, all failed in the long run because, as Axhausen and others have shown, of absorption or extrusion of the iso- and homotransplants, and of contraction of the autotransplants, with the production in all cases of intractable strictures.

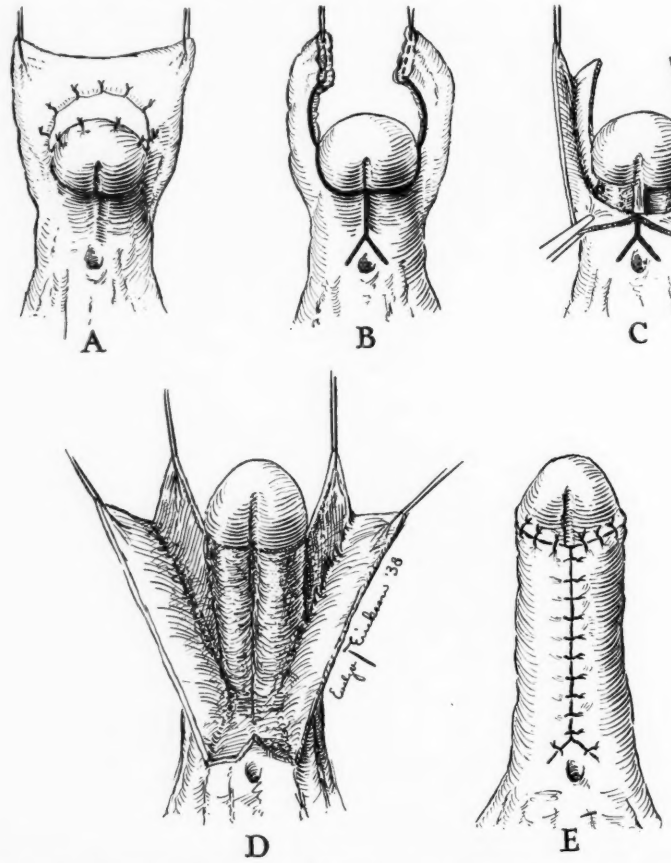


Fig. 2.—Edmunds' method of straightening the penis. *A*, First stage; "buttonholing" the foreskin; *B*, *C*, *D*, *E*, second stage; between steps *C* and *D* the fibrous remnant of the corpus spongiosum has been excised as in Fig. 1, *B* and *C*. (After McIndoe.[9])

The only one of these methods offering any hope of satisfactory results is that of Nové-Josserand as recently modified by McIndoe (Fig. 3). He sutures a Thiersch graft from the inner arm around a bougie having the diameter of the proposed urethra and inserts it within a special trocar, with which a tunnel is made from the tip of the glans to the old urinary meatus. The strictures which followed the original operation of Nové-Josserand are said by McIndoe to be prevented by leaving a

catheter in the new skin tube for a minimum of six months after its construction, before connecting it to the proximal urethra. Provided strictures do not form later, this is undoubtedly the simplest method of constructing the urethra.

The trouble and inconvenience of keeping a tube in the new urethra for so long a time cannot be lightly dismissed, particularly in the case of young children; the necessity for two operations in every case is also disadvantageous.

With this exception, all successful operations for the construction of the urethra have utilized pedicle flaps left attached throughout their

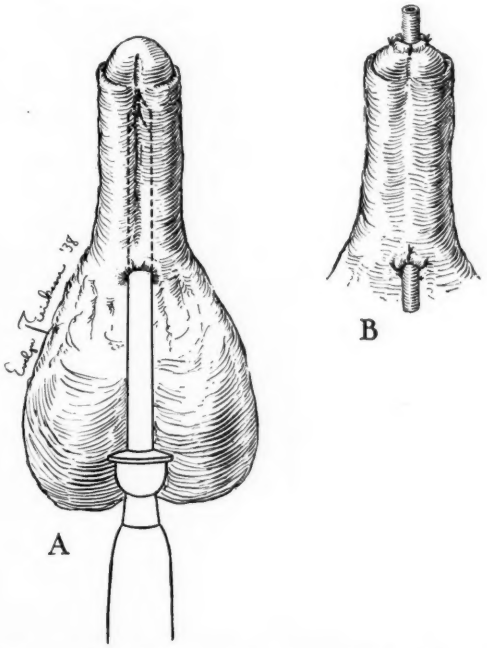


Fig. 3.—McIndoe's modification of the urethroplasty of Nové-Josserand. The Thiersch graft, stitched around a catheter, is contained within the author's special trocar which is shown being inserted in *A*. In *B* the trocar has been removed and the graft has been sutured to the edges of the tunnel. The catheter remains in situ. (After McIndoe.[5])

whole length and separated from their beds only at the edges. The forerunner of all these was the operation of Duplay in which the new urethra was formed by making a longitudinal incision in the ventral skin of the penis on each side of the midline, freeing the lateral edges of the long island of skin thus formed, and suturing them together, skin surface inward, over a catheter. The edges of the skin lateral to the tube thereby constructed were then united over it. This operation has been abandoned because the suture line in the new urethra lies immediately beneath that in the outer skin and fistulae rarely fail to develop.

The operation of Thiersch (1869) has overcome this objection by "staggering" the suture lines (Fig. 4). The first incision on the ventral surface of the penis is made almost in the median line, and the second lateral to it on the other side of the midline, so that the suture line in the new urethra lies nearly in the midline, close to its roof, and in contact with the corpus cavernosum of one side. This urethral flap must be of generous thickness. The outer flap (on the side of the initial incision) is then pulled over to the opposite lateral surface of the penis, a step which involves pulling the skin on the dorsum of the penis medially, and is held there by penetrating the tunica albuginea of the corpora cavernosa with the skin sutures.

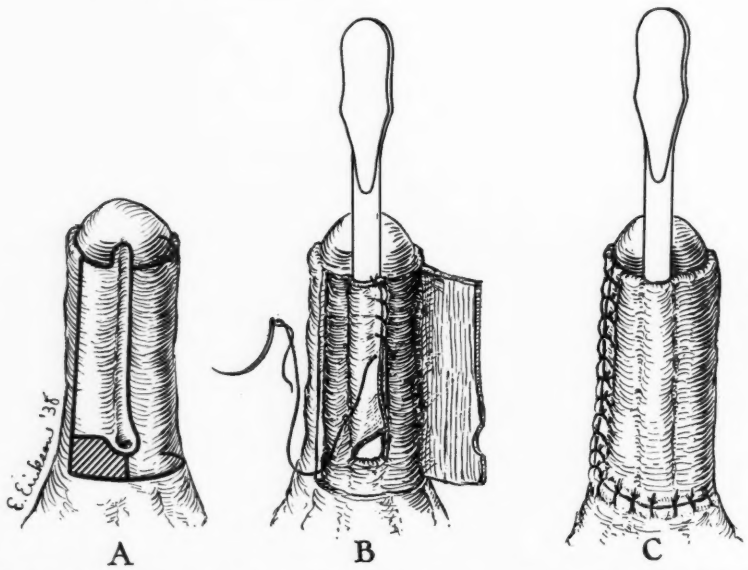


Fig. 4.—Modified Thiersch-Cecil urethroplasty. *A*, Incisions outlined; the cross-hatched skin is to be excised; *B*, construction of urethra; *C*, closure; note how the inner and outer suture lines are kept apart.

The two suture lines having been held so far apart, fistulae cannot form except as the result of a hematoma or abscess beneath the flaps. The former can be prevented by careful hemostasis, and the latter usually, but not always, by careful asepsis, since the skin of the penis and scrotum is difficult to sterilize.

A danger point, so far as fistula formation is concerned, is the junction between the new urethra and the old. This danger is practically eliminated if one overlaps the suture lines here sufficiently by excising a piece of skin one-half inch or more in length just behind the new meatus so that the suture line in the outer flap is well behind the transverse line at the commencement of the new urethra.

The imperative necessity for separating the suture line in the urethra from that in the outer skin by at least one-half inch, and preferably

more, has never been sufficiently emphasized as a preventive of fistula formation, a fact which is probably largely responsible for the continuous efforts still going on to develop "new" operations.

Most textbooks recommend that the urethra be fashioned over a catheter which is left in place until the wound heals, but I have preferred to use a sound (size 24 French) and to remove it after completion of the operation, both because its smooth surface permits withdrawal with less trauma and because, like Cecil, I believe that healing is more prompt and complete in the absence of a foreign body.

Many surgeons specify nonabsorbable sutures but, as Cecil has shown, abscesses may form about them, perforate the skin, and cause fistulae. The 000 chromic catgut preferred by him has proved far more satisfactory. I employ a continuous suture in the urethra to minimize the amount of catgut buried and interrupted sutures in the outer skin.

It may be objected that this operation does not bring the urethra out through the glans, but creates a glandular hypospadias. This is true, but constitutes no objection, since the resultant deformity is apparent only to a critical observer and causes no difficulty of any kind. Various supplementary operations may be used to bring the urethra through the glans, but there is danger of a stricture of the new meatus which does not follow the operation just described.

In perineal hypospadias, which is uncommon, it may be necessary to use hair-bearing skin for the posterior part of the new urethra. The danger that the hair will continue to grow and will become encrusted with urinary salts may be eliminated by destroying the hair follicles prior to operation with diathermy, although McIndoe's method would appear to be preferable here.

From the viewpoint of fistula formation, the operation of Bucknall (1907) is most nearly foolproof, but it has been largely abandoned because the floor of the urethra is formed from hair-bearing scrotal skin (Fig. 5). Cabot and others have found that this hair continues to grow and have observed stone formation upon it. Moreover, the operation is not feasible for perineal or penoscrotal hypospadias when the scrotum is small.

Cabot has recently overcome the first objection by constructing the urethra itself from penile skin after the manner of Thiersch and then embedding it in a trough in the scrotum until healing has occurred (Fig. 6). It is then freed and the raw area covered, as in Bucknall's method, by lateral pedicle flaps of scrotal skin. This method is useful when the urinary meatus is distal to the penoscrotal angle and the scrotum well developed.

It should be noted that both Cabot's and Bucknall's methods involve two operations for the construction and covering of the urethra. That

of Thiersch ordinarily requires but one session; a second operation upon the urethra will be required only if fistulae develop.

Another operation which has enjoyed a definite vogue is that of Ombredanne. The urethra is constructed by a pedicle flap of skin taken from the ventral surface of the penis behind the meatus, to the posterior

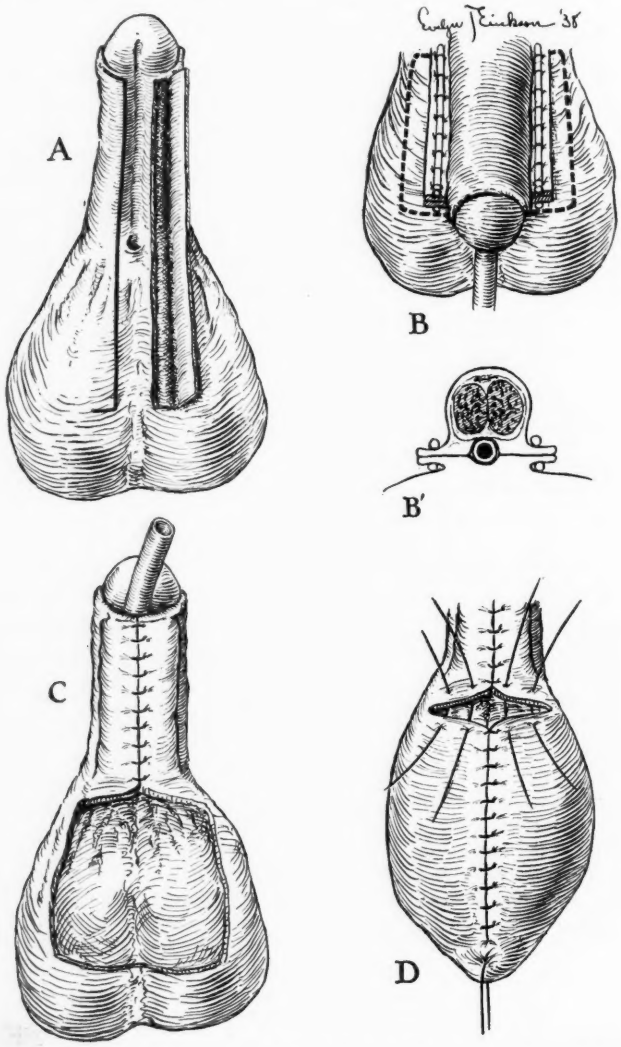


Fig. 5.—Bucknall's operation. *A, B,* First stage; *B',* cross-section of *B.* The dotted lines outline the scrotal flaps which will be liberated at the second stage and used to cover the new urethra in *C. D,* Closure of defect in scrotum. (After Campbell, M.: Pediatric Urology, 1937, New York, The Macmillan Company.)

edge of which it is left attached. This flap is turned forward by means of a purse-string suture so that its anterior end reaches the inferior surface of the glans, where it forms the new meatus. The raw surface thus left is covered by buttonholing the foreskin and bringing it down

over the ventral surface of the penis. The two lateral pedicles are divided later. This operation is said to obviate fistula formation but it leaves a pouch-like urethra, the dribbling from which after micturition is likely to be very inconvenient for the patient. This is especially true in those cases in which several stages are required to bring the urethra to the end of the penis, leaving a series of pouches, each of which may retain a little urine.

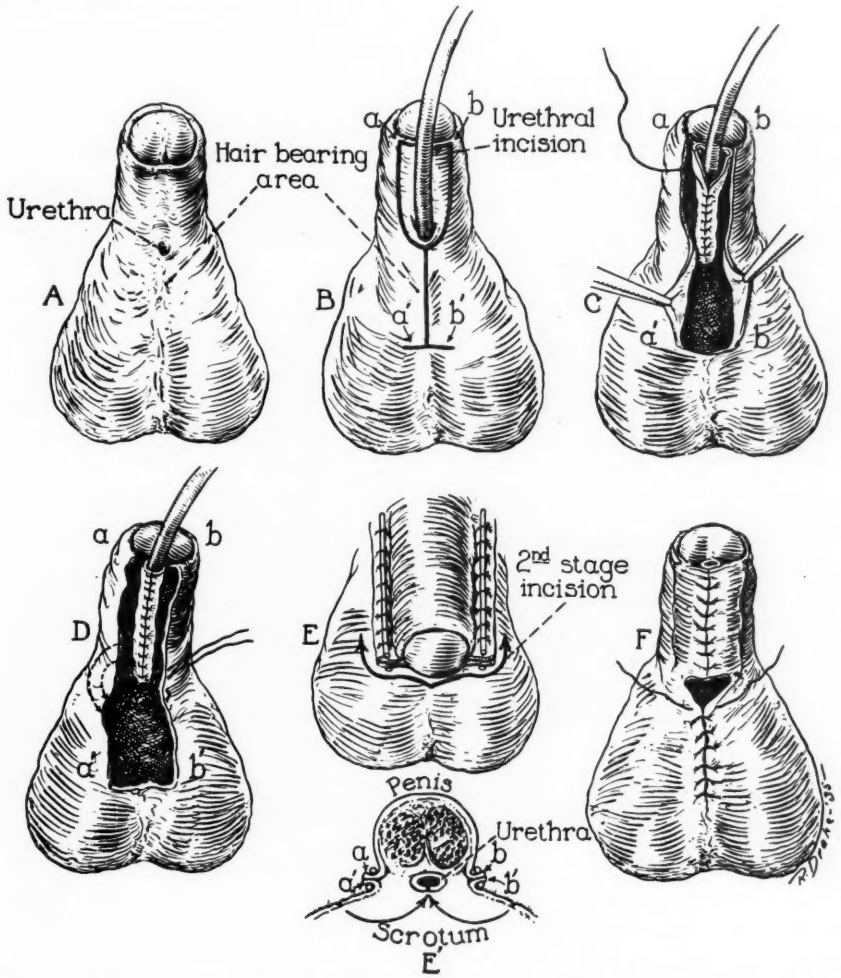


Fig. 6.—Cabot's modification of Bucknall's method. (Courtesy of Dr. Hugh Cabot.)

Everything considered, I believe that the operation of Thiersch is most practicable and is freest from objectionable features if the necessary care in its performance is employed.

RESULTS

An unexpected tendency to fistula formation at the site of perineal urethrostomy must be mentioned. This method of diverting the urine

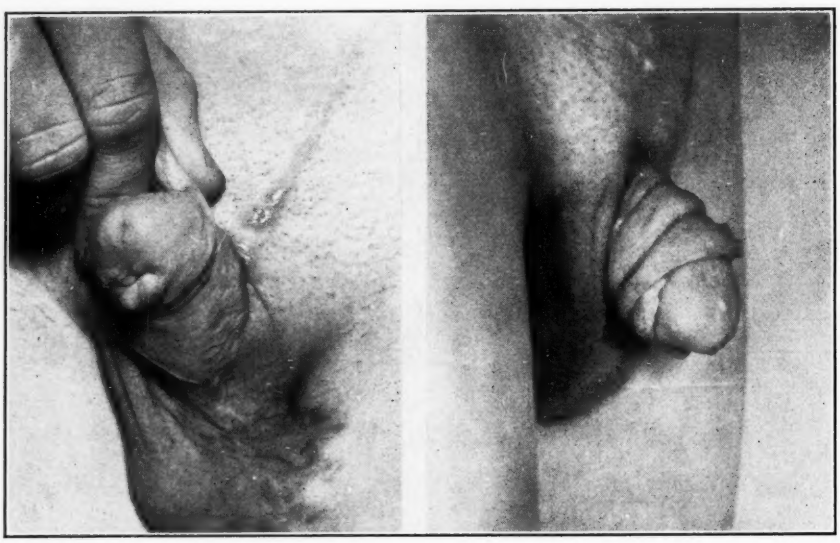
was used 14 times in 13 cases in connection with straightening of the penis and 10 times in 9 cases in connection with urethroplasty, a total of 24 times.

Fistulae followed in 3 (Table I). In one (R. D.) this was due to the fact that an encircling ligature of silkworm gut was inadvertently left buried in the perineum. Prompt healing followed its removal.

In the second (D. F.) the fistula was small and healed after excision under local anesthesia in January, 1938, without hospitalization.

In the third (L. C.) too little tissue intervened between the urethra and the surface. It was closed by overlapping flaps in December, 1937.

The penis has been straightened in 13 cases by the method of Duplay supplemented, when necessary, by separation of the urethra from the



A. *B.*

Fig. 7.—*A*, Anterior and *B*, oblique view of penis of N. M., made 28 months after straightening the penis by Duplay's method, 18 months after a Thiersch-Cecil urethroplasty, and 6 months after orchiopexy.

corpora and posterior displacement of the meatus. The result was perfectly satisfactory in all but one (J. St.), in whom the deformity had recurred six months later when he returned for urethroplasty. Recurrence was due to contracture of the operative scar and was corrected by excising the scar, freeing the urethra from the penis, and allowing it to retract to a new position. A perfectly straight penis resulted.

Urethroplasty has been done in 9 cases, 4 of the 13 already straightened not having returned as yet for the second operation.

In the first case (H. M.) three minute fistulae resulted from a Duplay urethroplasty done because I did not appreciate that the suture lines must be kept away from one another. The patient has refused to permit closure of the fistulae.

TABLE I

RESULTS OF THIERSCH-CECIL URETHROPLASTY

CASE	AGE	DATE OF OPERATIONS			COMMENT	RESULT
		STRAIGHTENING	URETHROPLASTY	OTHER		
J. Sh.	15	Oct. 25, 1930.	Did not return	----------	----------	Straight penis
H. M.	15	Sept. 14, 1931.	March 22, 1933 (Duplay)	----------	Three small fistulae; patient refused reoperation	Three fistulae
L. C.*	18	April 12, 1933.	May 17, 1934 (Duplay) broke down	Repeated April 3, 1936; left four fistulae	Closure fistulae March 5, 1937; reclosure Dec. 28, 1937	Good
J. St.	9	Aug. 28, 1933, and Jan. 18, 1934.	To be done later	----------	Recurrence of curvature, reoperation	Straight penis
L. M.	16	Nov. 29, 1934.	Aug. 5, 1935	Closure fistula Oct. 1, 1936	Small fistula; operative closure	Good
N. M.	14	Aug. 2, 1935.	June 17, 1936	Orchiopexy July 20, 1937	None	Good
A. M.	6	Jan. 30, 1936.	July 3, 1936	----------	None	Good
L. J.	7	March 2, 1936.	July 26, 1937	Closure fistula Jan. 6, 1938		Good
D. F.	28	July 14, 1936.	June 22, 1937	Closure perineal fistula Jan. 2, 1938	Fistula closed without hospitalization	Good
B. S.	11	July 17, 1936.	June 18, 1937	----------	None	Good
R. D.	8	July 29, 1936.	July 13, 1937	----------	Fistula at urethrostomy closed spontaneously after removal suture	Good
W. G.	12	Aug. 28, 1936.	To be done later	----------	----------	Straight penis
M. B.	5	Sept. 15, 1936.	To be done later	----------	Stricture hypospadiac meatus	Straight penis

*See footnote on page 730.

In the second (L. C.)* a repair of the Duplay type broke down and had to be repeated, following which two operations to close fistulae were required. The end-result is good nonetheless. I have since abandoned this operation for that of Thiersch.

In the succeeding 7 cases there have been 3 fistulae. The first of these (N. M.), although very small, was closed by overlapping flaps at a secondary operation. The second (L. J.) developed at the penoscrotal angle because of insufficient staggering of the suture lines at this point, and was closed without diversion of the urine in January, 1938. The third (D. F.) followed incision of an infected hematoma but closed of itself before the patient left the hospital.

To summarize, urethral fistulae followed both the Duplay operations; the Thiersch-Cecil urethroplasty has been followed by fistulae in but 2 of 7 cases. That this is an objection to the method cannot be denied; when one considers that the methods alleged to be less likely to result in fistulae formation (operations of Ombredanne, Cabot, McIndoe) customarily require two stages, this objection is less serious, since only 2 of 7 cases have required a second operation.

SUMMARY AND CONCLUSIONS

1. Glandular hypospadias does not require treatment save in exceptional cases.

2. The penile, penoscrotal, and perineal varieties require surgical correction to remove the attendant sexual disability and the deformity.

3. The ventral curvature of the penis (congenital chordee) must first be corrected.

4. Of the many methods devised for bringing the urethral tube to the end of the penis, the method of Thiersch with the important modifications of Cecil seems most satisfactory for general use.

5. Cabot's combination of the method of Thiersch with that of Bucknall has a definite field of usefulness.

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*This patient deserves separate mention. He was a masculine complete pseudohermaphrodite with perineal hypospadias and undescended testes. He had a rudimentary uterus and vagina which, together with the urethra, opened into a persistent urogenital sinus in the perineum. Abdominal exploration disclosed rudimentary gonads in the position of ovaries; biopsy revealed immature testes.

The penis was straightened by Duplay's method in February, 1933. A Duplay urethroplasty in May, 1934, broke down completely. It was repeated in April, 1936, and left four fistulae. These were closed by overlapping flaps in March, 1937. Three of the fistulae healed, but the fourth recurred, now of filiform size, and a fistula remained at the site of the perineal urethrostomy. These were closed by overlapping flaps in December, 1937.

As if these misadventures were not enough, the patient developed a stricture of the new meatus after the second urethroplasty, at which most of the urethra was constructed by Duplay's operation, while Young's method was used to bring the urethra to the end of the penis. That repeated dilatation by the patient has resulted in a satisfactory meatus in the normal situation is a tribute to his excellent cooperation and dogged persistence.

Whether this is an example of commendable persistence or of farcical bungling I leave the reader to decide. The lessons learned at this patient's expense have undoubtedly reacted to my benefit and to that of the subsequent patients.

The multiple failures were unquestionably due to my slow awakening to the fact that the suture line in the outer skin must be as far as possible from that in the urethra. The importance of this fact cannot be overemphasized.

The occurrence of the stricture at the new meatus has led me to abandon the effort to place the meatus at the tip of the glans because it does not seem worth the inconvenience to the patient.

6. McIndoe's modification of the operation of Nové-Josserand may prove very valuable.

7. The results of the Duplay operation for straightening the penis are reported in 13 cases. The importance of freeing the urethra from the corpora cavernosa and allowing it to slip back has been emphasized.

8. Duplay's operation for forming the urethra has been abandoned because multiple fistulae followed its use 3 times in 2 patients.

9. The results of the operation of Thiersch and Cecil are reported in 7 cases. Two (28½ per cent) were followed by fistulae which required operative correction.

10. Thus the urethra has been constructed by Thiersch's method in one stage in 71.5 per cent; most methods regularly require two stages.

11. One postoperative stricture developed. It involved the external meatus in the only case in which Young's operation was used to bring the urethra through the glans.

REFERENCES

1. Axhausen, G.: Prognosis of Transplantation of the Appendix in Hypospadias, Berl. klin. Wchnschr. **55**: 1065, 1918.
2. Bucknall, R. T. H.: A New Operation for Penile Hypospadias, Lancet **2**: 887, 1907.
3. Cabot, H.: The Treatment of Hypospadias in Theory and Practice, New England J. Med. **214**: 871, 1936.
4. Cecil, A. B.: Surgery of Hypospadias and Epispadias in the Male, Tr. Am. A. Genito-Urin. Surgeons **43**: 253, 1931.
5. Duplay, S.: De l'hypospadias perineo-scrotal et de son traitement chirurgical, Arch. gen. de med. de Paris **23**: 513, 1874.
6. Edmunds, A.: Surgical Treatment of Pseudohermaphroditism and Hypospadias, Lancet **1**: 323, 1926.
7. Legueu, F.: Repair of Urethral Defects by Tubular Grafts of Vaginal Mucosa, J. Urol. **2**: 369, 1918.
8. Lesser, E.: Beitrag zur Vererbung der Hypospadie, Virchow's Arch. f. path. Anat. **116**: 537, 1889.
9. McIndoe, A. H.: The Treatment of Hypospadias, Am. J. Surg. **38**: 176, 1937.
10. Mettauer, J. P.: Practical Observations on Those Malformations of the Male Urethra Termed Hypospadias, Am. J. Med. Sc. **4**: 43, 1842.
11. Nové-Josserand, G.: Traitement de l'hypospadias, Lyon med. **85**: 198, 1897.
12. Ombredanne: Penile Hypospadias in an Infant, Bull. et mém. Soc. de chir. de Par. **57**: 1076, 1911.
13. Schmieden, V.: New Method of Operation for Male Hypospadias, Arch. f. klin. chir. **90**: 748, 1909.
14. Thiersch, C.: On the Development and Surgical Treatment of Epispadias, Arch. d. Heilkunde **1**: 20, 1869.
15. Tuffier: Treatment of Hypospadias, Ann. d. Mal. d'Org. G. U. **17**: 370, 1899.
16. Weitz, H.: Treatment of Hypospadias, Deutsche med. Wchnschr. **41**: 1064, 1915.
17. Young, H. H., and Davis, D. M.: Practice of Urology, Philadelphia, 1926. W. B. Saunders Co., Vol. 2, p. 603.

TREATMENT OF DISLOCATION OF THE SHOULDER

HENRY MILCH, M.D., NEW YORK, N. Y.

IN THE numerous articles which have appeared upon the subject of dislocation of the shoulder, practically every conceivable form of therapy, conservative, manipulative, or radical surgery, has been described. Many of the procedures suggested have only limited application to special problems, such as the chronic dislocation, the recurrent dislocation, or the dislocation associated with fracture. In general they do not apply to the handling of the relatively acute dislocation. In the routine treatment of the acute, anterior subcoracoid type of dislocation, too, many different methods have been proposed, but all have been largely superseded by the manipulative method of Kocher. As a result, the chance application of any of the other methods is often greeted with complete astonishment, even by competent surgeons.

Though Kocher's manipulative method undoubtedly represented a great advance over the older, more brutal traction and leverage methods, it is not entirely devoid of danger. Stimson[6] specifically warns that "breaking the humerus during the second step—outward rotation—must be borne in mind, especially in elderly patients." However, it must not be believed that this contingency arises only in the elderly, as the following case demonstrates.

CASE REPORT.—Gus M., a robust laborer, in his early twenties, was seen on Nov. 3, 1936, following an unsuccessful attempt at Kocher reduction of a dislocation of the right shoulder. The dislocation had occurred some forty-eight hours earlier as a result of a fall upon the shoulder. The attempt at reduction under anesthesia was followed by a snapping sound, and x-ray subsequently disclosed a long, spiral fracture of the upper third of the humerus. The short, medial fragment was disclosed in the subcoracoid portion. The larger fragment was rotated externally and was itself broken in two fragments, which were angulated, in a manner suggesting that the smaller fragment, comprising a portion of the great tuberosity, was held fixed by the supraspinatus muscle (Fig. 1).

Further manipulative procedures were considered inadvisable and open operation was performed. The long half of the biceps was found lacerated and lay in the cleft of the spiral fracture, firmly binding the medial fragment. The lacerated tendon was cut away and the medial fragment was dislodged with difficulty, after tenotomizing the pectoralis major, the coracobrachialis, the short head of the biceps, the latissimus dorsi, subcapillaries, and finally the medial portion of the capsule. Only then could reposition be accomplished. Following reduction, the fractured fragments were sutured with kangaroo tendon; the tenotomized muscles were sutured and the skin was closed. The patient was treated in abduction with traction. Uneventful convalescence, with ultimate return of moderate function, rewarded these efforts.

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As this case was reviewed, it seemed that the clinical findings could be explained only by assuming that the head, fixed in internal rotation by the biceps or the internal rotators, had been split in a spiral manner from the shaft during the maneuver of external rotation. The importance of the biceps in preventing reduction had previously been noted by Cubbins, Callahan, and Scuderi.[1] They state: "As nearly as we have been able to determine . . . these dislocations are caused first and most commonly by the firm hold of the long head of the biceps. . . . The

Fig. 1.—Spiral comminuted fracture of dislocated right humerus, resulting from an attempt at Kocher manipulation.

head of the biceps simply holds the humeral head firmly until the former has been freed from its groove or slipped forward over the humeral head, when out of the groove. But when the head of the biceps is released, the humeral head, as a rule, can be freed immediately." This contingency does not appear to be envisaged in the usual explanation of the mechanism of Kocher's manipulative reduction. Indeed, some authors categorically deny the importance of the muscles in the mechanism of reduction. Stimson quotes Ceppi to the effect that external rotation

served to unwind the upper part of the capsule and the coracohumeral ligament. He further observes that: "External rotation during traction was first employed under the influence of various ideas concerning the part taken by the muscles in opposing the return of the bone . . . then in the light of more accurate knowledge of the influence of the untorn portion of the capsule, it became the first step in the methods of reduction without traction." Speed,[4] on the other hand, states that "the muscular resistance of the subscapularis and pectoralis is overcome by steady outward rotation in the first stage of the reduction." The contradictory statements of these two eminent authorities can only be reconciled, if at all, on the presumption that one was primarily interested in the mechanism of the reposition, while the other was concerned principally in explaining the steps preliminary to the actual act of reduction. But even Speed stresses only the action of the pectoralis and the subscapularis. No mention, however, is made of any effort to relax the other muscles about the shoulder joint. It was largely to overcome their actions, particularly that of the biceps, that the operation performed by Cubbins and his associates was suggested in the treatment of old and irreducible dislocations.

Despite these contrary views, it is undoubtedly the opinion of those who have had an extensive experience that muscular action plays a large part in preventing the reduction of even acute dislocations. This is amply attested to by the ease with which, under general anesthesia, many dislocations, previously irreducible, yield after relaxation of the tense, surrounding muscles. That the muscles involved include not merely the pectoralis, the subscapularis, and the biceps is evidenced by the fact that many apparently simple dislocations are complicated by avulsion fractures of the great tuberosity, indicating participation of the external rotator muscles. In different manner and degree all the shoulder muscles must exert their forces on the dislocated as well as on the normally placed humeral head. Indeed, even allowing for their natural elasticity, it is hardly conceivable that muscles, adapted to a normal position, should not exert incalculable abnormal stresses in the suddenly altered relationship between the humeral head and the glenoid cavity.

A casual review of the course of the perihumeral musculature emphasizes the apparently haphazard manner in which these muscles appear to act upon the shoulder joint when in the anatomic position (Fig. 2). Some of the muscles run upward and outward, some downward and outward, others up and down, and still others simply outward. In this position their actions are in many instances completely antagonistic. However, the anatomic position is not the usual position of function of the arm. In each of the other positions which the humerus assumes the interplay of muscles and, in consequence, the resultant of their actions varies, depending on the relationship existing between their individual direction and the axis of the humeral shaft.

Though it is not intended to enter any controversy on the complicated kinesiology of the shoulder joint to which Codman and others have called attention, it must be evident that an important factor in determining this resultant of forces will be the position of the humerus in relation to the scapula. Contrary to the general belief that only a small part of the shoulder motion occurs in the glenohumeral joint, Steindler,[5] in discussing abduction of the shoulder, states that "this entire excursion field is 110 to 120 degrees." X-ray plates, with the arm at the side and in complete abduction, readily demonstrate the validity of the con-

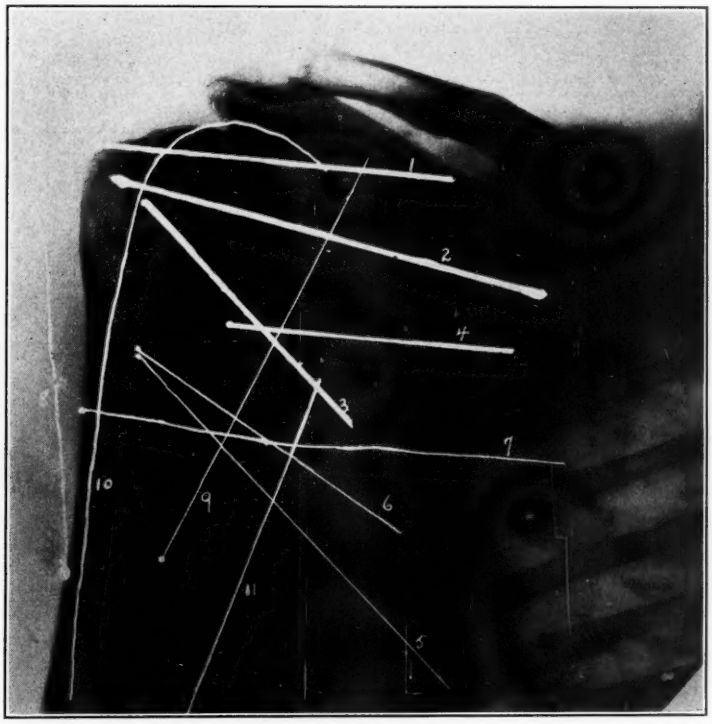

Fig. 2.—Scheme showing the projected course of the perihumeral muscles, with the normal arm at the side. Note the angle which the adducted humerus makes with the axillary border of the scapula and the manner in which the lines of action of the various muscles cross one another.

clusion. It will be observed that in the abducted position the axis of the humeral shaft is roughly parallel with the midline of the scapula and may even be brought into alignment with the axillary border of this bone (Fig. 3). In the overhead position, the position which phylogenetically characterizes the era of arboreal brachiation, all the muscles assume a marked symmetry of disposition. It will be seen that the muscles about the shoulder may be divided into three groups, each of which may be considered as lying upon the surfaces of three more or less conical figures. The first group consists of the supraspinatus, the infraspinatus, the teres minor, and the subscapularis which converge on the

apex of a small, inner cone to form the musculotendinous cuff described by Codman. The second group is made up of the pectoralis major, the latissimus dorsi, and the teres major. The apex of this larger and more

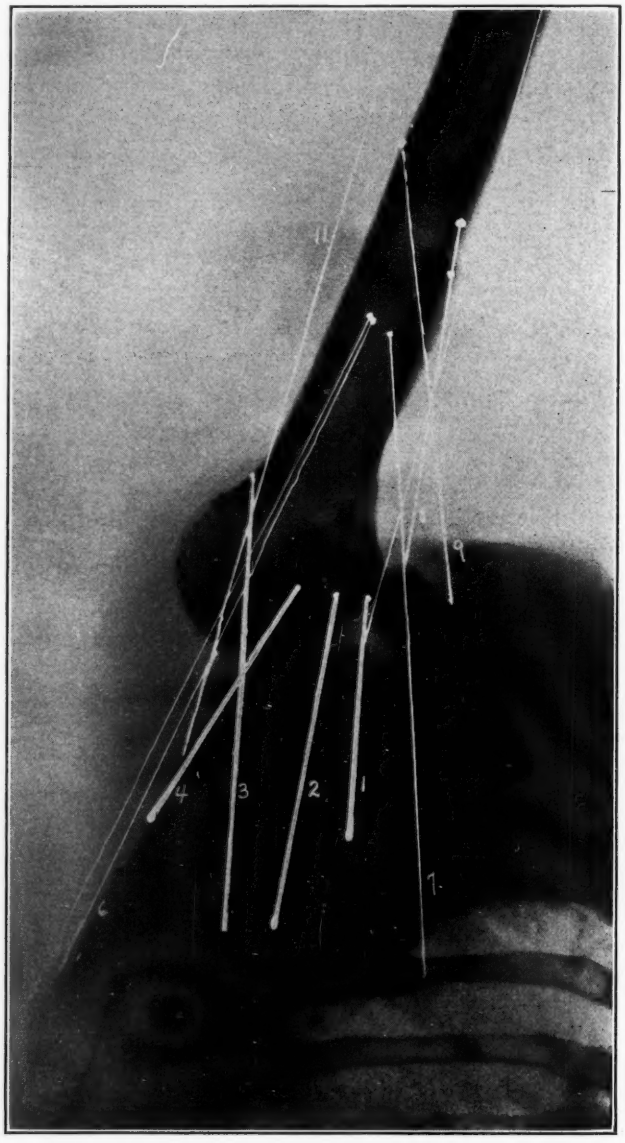

Fig. 3.—The same arm in complete, overhead abduction. Note the change in relationship of humerus to scapula and of scapula to thoracic wall. Note the conical symmetry of disposition of the perihumeral muscles, with the apices of the cones lying on the humeral axis. In this position the resultant of force of all the muscles about the shoulder lies in a line which is parallel to and frequently collinear with the shaft of the humerus.

superficially situated cone is more distally placed on the shaft of the humerus. The third group, consisting of the deltoid, the coracobrachialis, the biceps, and the triceps, form a cone whose apex is still more dis-

tally situated, when the elbow is extended. (Even though the deltoid and the coracobrachialis do not insert at the level of the other two muscles, they may be grouped with them from a kinetic point of view.) The apex of each of these cones is on the humerus and in the abducted position their axes are in general collinear with each other, with the shaft of the humerus, and with the midline of the scapula. In other words, the overhead position is the one in which the cross-stresses due to the oblique course of the different muscles are absent or are reduced to a minimum. It was in all likelihood the recognition of these facts that formed the basis of the method of reduction by upward traction. Originally attributed to Celsus and subsequently redescribed by Duplay, Malgaigne, Mothe, Bransley, Cooper, and others, the method unfortunately fell into disuse, because of the fear of doing additional injury to the vessels and nerves.

While it must be admitted that even the judicious application of traction may result in damage to the brachial vessels and nerves, great care must be exercised in determining the etiology of the nerve lesions. In a case which I reported some years ago[2] an extensive brachial palsy, involving the whole arm, occurred primarily as a result of the dislocation and was noted before any attempt at reduction. At that time the remark was made that the number of nerve lesions associated with simple dislocation of the shoulder was probably small. Since that time, a number of additional articles stressing this concomitance have appeared, and my original opinion as to the relative infrequency of this complication has been completely reversed.

Through the courtesy of various members of the attending staff at the Hospital for Joint Diseases, I was given the privilege of reading the charts of a number of patients treated for dislocation of the shoulder. Of a series of 24 in-patients treated for dislocation of the shoulder within the past four years, 7 showed associated nerve lesions and 9, fractures of the greater tuberosity. In 2 of these cases both fracture and nerve lesion occurred in the same patient. Since a number of cases of simple dislocation were treated in the Out-Patient Department during the same period, these figures were considered of value only in establishing the relative frequency of fractures as compared with nerve injuries in cases of dislocated shoulder. It appears that in the more severe types of dislocation nerve lesions occur only slightly less often than the more commonly recognized fractures of the greater tuberosity. (It is possible that more careful examination of injured patients, prior to reduction, with particular attention to nerve complications, may further document this general statement. In any event, it indicates the care with which conclusions as to danger inherent in any procedure must be scrutinized.) In most cases the nerve lesion was noted on the chart as being present before the reduction, usually the Kocher manipulation, was attempted.

Although there was no uniformity of nerve involvement, it is interesting to note that the axillary nerve was affected twice, the radial once, and the ulnar four times. The greater preponderance of ulnar nerve paralyses seemed to suggest the analogy between the mechanism of their production by stretching and that noted in the case of Erb's paralysis, cervical rib, or first dorsal rib simulating cervical rib.[3] It served also to suggest that the method of upward traction should be modified by the avoidance of any additional stretching force.

At present, no traction whatsoever is employed in the method of reduction, although the overhead position is retained. Counter-traction or fixation of the scapula has been omitted. The patient lies in the supine position, while the surgeon takes his position on the side of the dislocation. In a right sided dislocation the surgeon places his right hand upon the patient's right shoulder, so that the fingers find firm support on the top of the shoulder, while the thumb is braced against the dislocated humeral head (Fig. 4*A*). The right hand fixes the head as the left hand gently abducts the arm into the overhead position (Fig. 4*B*). During this maneuvre the head of the humerus is supported so that it cannot move from its dislocated position. As a consequence, instead of moving downward as the arm moves upward, the head rotates in place. In this manner the tendency to further stretch already stressed nerves is obviated. As the arm is abducted, it is gently externally rotated to release the twisted capsule, according to the principles described by Kocher. Once the arm has been brought into complete abduction in this overhead position, all cross-stresses exerted by all the muscles have been eliminated; the head can be gently pushed over the rim of the glenoid and the dislocation reduced (Fig. 4*C*). Even in old and nervous patients these maneuvers may be, and have been, successfully carried out, without the use of an anesthetic. This can be done only providing the surgeon has the patient's confidence and does not abuse it by causing excessive or unnecessary pain. Where this manipulation cannot be carried out, a few whiffs of gas usually suffice to provide sufficient muscular relaxation to permit ready reduction.

The method which may be called that of abduction, external rotation, and pulsion has been employed, with some modifications, for a number of years and has given complete satisfaction. No bone fracture and no nerve lesions attributable to its use have been observed. It has the distinct advantage that in suitable cases it may be used as an out-patient procedure, without any form of narcosis. Where it can be used without anesthesia, prolonged hospitalization and the danger of postanesthetic complications may be avoided. The method has been used mainly in the subcoracoid type of dislocation. It has been used in one case of supraglenoid dislocation in which reduction occurred immediately upon elevating the arm.

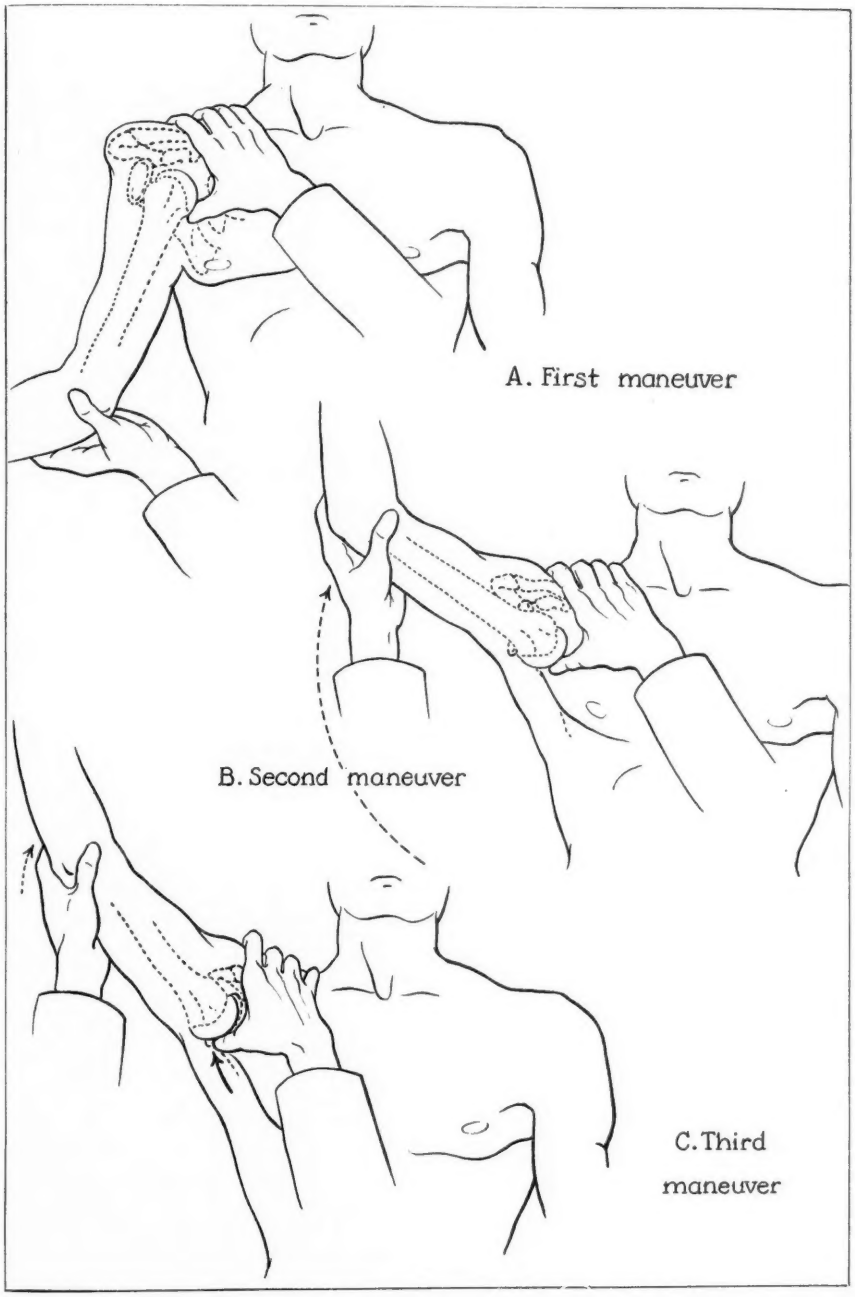

Fig. 4.—*A*, First maneuver: the patient supine, the surgeon's right hand is braced against the shoulder, while the thumb fixes the humeral head; *B*, second maneuver: the patient's dislocated arm is abducted in external rotation, the thumb pressure against the humeral head is still maintained; *C*, third maneuver: with the arm in the completely abducted position, the surgeon's thumb gently pushes the humeral head over the glenoid ridge into the fossa.

Though this procedure has found its greatest application in the routine handling of uncomplicated dislocations, it may prove of service in the treatment of fracture dislocations of the upper end of the humerus. In a case recently reported to me by one of my colleagues, it was successfully employed in a typical fracture dislocation for which open reduction had been advised. While our experience is not sufficient to justify any statement as to the value of the method in this class of cases, it would seem justifiable to attempt the maneuvers described before establishing the more serious indication for operative correction.

REFERENCES

1. Cubbins, W. R., Callahan, J. J., and Scuderi, C. S.: Surg., Gynec. & Obst. **58**: 129, 1934.
2. Milch, Henry: Am. J. Surg. **5**: 66, 1928.
3. Milch, Henry, and Brickner, W.: Surg., Gynec. & Obst. **40**: 38, 1925.
4. Speed, K.: Fractures and Dislocations, New York, 1916, Lea & Febiger, p. 423.
5. Steindler, A.: Mechanics of Normal and Pathological Locomotion in Man, Springfield, Ill., 1935, Charles C. Thomas, p. 302.
6. Stimson, L. A.: Fractures and Dislocations, Ed. 7, New York, 1912, Lea & Febiger, p. 636.

CONGENITAL AND DEVELOPMENTAL COXA VARA

GEORGE A. DUNCAN, M.D., SC.D. (MED.), NORFOLK, VA.
(From the New York Orthopaedic Dispensary and Hospital)

THE purpose of this paper is to report 31 cases of congenital and infantile coxa vara and to analyze their treatment. Coxa vara is a diminution in the angle of inclination of the femoral head and neck with its shaft from the normal of 120 to 130°. It is not a rare condition for frequent reports are found in the literature, nor is it so common that there is a unanimity of opinion concerning its etiology, diagnosis, and treatment.

Fairbanks,[1] modifying Elmslie's classification, divides coxa vara into: (1) congenital, (2) infantile or cervical (developmental), (3) rachitic, (4) adolescent, (5) traumatic, (6) secondary to inflammatory troubles (e.g., tuberculosis of the femoral neck), (7) associated with local or general bone disease, such as fibrocystic disease, osteogenesis imperfecta, osteitis deformans, etc.

Only congenital and infantile coxa vara are considered in this paper. These two types are essentially different stages in the development of the same condition and present certain characteristic features in common.

In the congenital group I include those cases which are associated with some other congenital anomaly of the femur, such as congenital dislocation of the hip or deficient ossification. These disturbances range from a mild hypoplasia to extensive aplasia of the femur.

I prefer to designate this second type as developmental, rather than infantile, as I believe it is a more accurate term. The developmental coxa varas include those cases in which one or more fissures across the femoral neck are present, characterized roentgenographically by one or more clear transverse lines, other than the epiphyseal line. Elmslie and Fairbanks point out that this type is often characterized by separation of a triangular piece of the femoral neck, base inferiorly. The presence of this separate triangular piece is just a manifestation of the developmental error and is not a definite reason alone for the separate classification. In Fig. A there is a triangular piece of bone representing the inferior angle of the femoral neck while the angle of inclination of the femoral neck appears normal. This child aged 8 years, may develop coxa vara later but at the present time the triangular bony defect must be considered a part and not the cause of the developmental error. Coxa vara found after 4 years of age and without associated congenital deformities is developmental rather than congenital.

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As to the various other types listed in the classification, the third variety (rachitic) is not commonly found. A diagnosis can be made only when associated with other signs of rickets and when the femoral capital epiphysis exhibits the typical radiographic changes (broadening and distortion of the epiphyseal disk, irregular deposits of calcium, and general loss of bone density) and when active rickets were present after the patient began to stand and walk. The fourth variety, due to slipping of the upper femoral epiphysis during adolescence, and the last three varieties can hardly be confused with the types under discussion.

ANATOMY AND OSTEOGENESIS OF THE UPPER FEMUR

No center of ossification is present in the head of the femur until the end of the first year after birth. The cartilaginous neck, which is very short, is in normal coxa valga, and the cartilaginous head therefore sits almost directly on the shaft of the femur. Between the third and fourth years the upper portion of the femoral shaft which corresponds to the

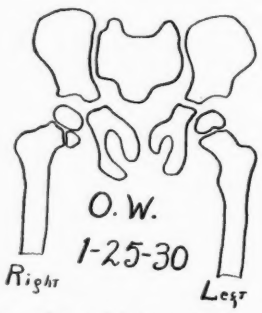


Fig. A.—Tracing of the x-ray of a patient, aged 8 years, with a separate triangular fragment of bone at the inferior angle of the femoral neck. No coxa vara was present. The child has been walking for several years.

femoral neck expands upward. During the fourth year, the center of ossification for the greater trochanter is formed. The capital epiphysis at this time rests on the entire width of the oblique femoral neck. The femoral neck, which at first was cartilaginous, is now composed of bone with hyaline cartilage covering its superior surface. This cartilage is continuous with the capital epiphyseal plate and the cartilage encircling the center of ossification at the site of the greater trochanter. Due to the normal angle of 130° assumed by the head and neck of the femur, the epiphyseal line is oblique. Because of this obliquity, the inferior angle of the femoral neck juts out or shows a slight prominence. This promontory acts as a buttress preventing the capital epiphysis from slipping downward. Sutro[2] has used this anatomic description to explain the mechanism of the capital epiphysis slipping in adolescence.

ETIOLOGY AND PATHOLOGY

Various theories regarding the etiology of congenital and developmental coxa vara have been advanced but they are all purely hypo-

thetical. This is because the microscopic appearance of bone removed from the affected portion of the femoral neck shows nothing characteristic.

Specimens have been removed by Hoffa,[3] Jones,[4] Elmslie,[5] Delitala,[6] Nilsonne,[7] Camitz,[8] Barr,[9] and Zadek[10] and at the New York Orthopaedic Dispensary and Hospital, no two of which have been found to have the same abnormalities. Some have found epiphysis present; others have not; some have found islands of cartilage in the epiphysis; others have not; and still others have found islands of bone and connective tissue lying about in profusion. Therefore, with this lack of uniformity in the histologic findings, various etiologic hypotheses have been advanced.

Alsberg,[11] Hoffa,[3] Kredel,[12] and Reiner[13] thought that because of intrauterine pressure the neck of the femur loses its power of growth. Bohm[14] thought it was atavism and in support of this idea pointed out that in anthropoid apes the femoral neck is nearly at a right angle with the shaft of the femur. Fairbanks[1] disagreed with this view and the findings. Helbring[15] thought it was due to a delay or lack of proper ossification. Nilsonne[7] suggested that the anomaly of the neck of the femur may be due to an embryonic vascular disturbance and Noble and Hauser[16] agreed.

This vascular disturbance might upset the ossification of the femoral neck and the epiphyseal line. Such vascular disturbance may be of various grades. If severe, there may be a complete absence of the upper end of the femur. If moderate, and this is the usual type of disturbance, the inferior angle of the neck fails to ossify or it ossifies as a separate triangular piece of bone. In either case the normal buttress action of the inferior angle of the femoral neck for the capital epiphysis has been removed, which allows the femoral head to slip downward. This deformity usually appears after the child has reached weight-bearing age and tends to progress. This theory seems to offer the best anatomic-physiologic explanation of the deformity.

There are various other theories advanced to explain the cause of coxa vara; among these are: local inflammatory conditions, rickets, trauma, and developmental error. Coxa vara is so frequently bilateral that to assume it is due to local inflammatory conditions or trauma is unreasonable. When bilateral, the appearance of the hips is often the same. The upper femoral epiphysis in infants is quite stable, since forceful manipulations performed to reduce congenitally dislocated hips seldom causes a slipping of the upper femoral epiphysis. As for rickets, proof of rachitic changes are found uncommonly in coxa vara. Trauma is common in children. When a limp is first noticed, parents, patients, and even physicians often associate it with some trivial injury, when it may have existed long before this injury occurred. A traumatic history must be accepted with reservations if at all.

Developmental error therefore is the most logical explanation for this deformity. It is probably the result of delayed or failure of ossification in the upper epiphyseal disk. Two of the patients in this series form an excellent illustration of this theory and have been reported in a previous article.[17] They are identical twins, colored, aged 12 years, both with bilateral coxa vara, but each with the varus of the femoral neck marked only on one side (in one the right, and in the other the left hip). Roentgenograms of the hips of these two boys are mirror images of each other.

This finding of coxa vara in identical twins with the deformity in one being the mirror image of the other seems to add support to this theory of hypoplasia. It is only in the realm of coincidence that one could consider the causative factor in these two patients to be a local inflammatory reaction, rickets, or trauma. Rickets has been used to explain all deformities of bone of obscure pathology. While it is conceivable that these two patients may have had rickets, they certainly did not show any of the characteristic features of rickets when admitted to the hospital. Furthermore, it is hardly probable that rickets would have produced such a mirror image deformity in these two boys. Still another time-worn theory, birth injury, may be discarded by the same reasoning.

APPEARANCE OF THE DEFORMITY

The roentgen appearance is characterized by a depression of the femoral head and neck below the normal angle of 120 and 130°. The epiphyseal line is usually vertical and there is a loss of substance in the femoral neck at the epiphyseal disk. The epiphyseal disk, besides being vertical, may resemble an inverted Y with a small portion of bone between the two arms. Whether the epiphyseal disk is slightly oblique, vertical, or has the appearance of an inverted Y makes no difference, but this finding is the result of the amount of hypoplasia that has occurred. The loss of substance in the femoral neck may be of all gradations. To the uninformed, congenital or developmental coxa vara may be diagnosed a fracture because the wide defect gives at first the appearance of an ununited fracture. With increase in age, the trochanter may continue to grow and may come in contact with the ilium, while changes of an osteoarthritic nature occur in the acetabulum.

INCIDENCE, SIGNS, AND SYMPTOMS

The sexes were equally divided as a general rule; in this series the boys outnumbered the girls 18 to 13. Coxa vara seems to be slightly more common in the negro race, as 10 of the 31 patients are negroes; whereas, the percentage of all colored patients admitted to this clinic form a very small percentage of the total patients treated. The right hip was involved in 8, the left in 9, and both hips in 14 patients, making a total of 45 hips involved. The average age of these patients on ad-

mission was 8.5 years, the youngest being 1 year and the oldest 18 years. The average age at the time pathologic signs were noted by the parents or when the patient complained of aches or pains was 4.5 years.

The signs most commonly noted were a short lower extremity and a limp, possibly with a waddling type of gait. A short extremity was noticed by the parents soon after birth in 6 patients. In 13 patients the parents first noted something was wrong when the child started to walk and there was an unmistakable limp.

Of the most common symptoms, I found early fatigue, followed by aches and pains in the affected thigh on walking or running. After puberty the intensity of these symptoms may increase due to the deformity producing an osteoarthritis about the femoral head and acetabulum.

The severity of the limp was proportionate to the extent of the varus in the hip and shortening of the extremity. As the varus increases the mechanical advantage of the hip muscles decreases. The shortening averaged about 1 inch. The greater trochanter was higher than normal; there was an increase in lumbar lordosis and Trendelenburg's sign was frequently positive. Motions in the affected hip were reduced, particularly abduction, extension, and internal rotation. There was no muscle spasm or pains on moving the hip. There was no telescoping of the hip, either upward or downward, which differentiated this deformity from congenital dislocation of the hip.

TREATMENT

Congenital or developmental coxa vara once established usually tends to progression and there is increasing deformity and disability. Treatment should be directed toward obtaining and retaining correction of the deformity.

Earlier treatment recommended was abduction, either gradual or forceful, under anesthesia and its maintenance for a prolonged period without weight-bearing. This treatment was based on the hope that the correction would occur through the epiphyseal disk, but this seldom happens. Certainly the deformity should not progress during the period of recumbency, but to keep a child of 6 or 8 years of age in bed until their upper femoral epiphyseal line closes is unreasonable.

Noble and Hauser[16] stated "that operations on the femoral neck are as a rule disastrous on account of the very poor blood supply to this region. If an osteotomy is performed union is improbable." This I agree with and see no need of attacking the deformity at this site. Zadek[10] overcomes this nutritional disturbance by drilling holes in the neck of the femur and across the epiphyseal line, thereby opening up new blood channels and obtaining union of the epiphyseal disk. This operation is followed by a subtrochanteric osteotomy to correct the deformity.

Noble and Hauser further stated: "Subtrochanteric osteotomy does not attack the site of the disease, will be of temporary benefit only and will add complications to any future operation." They preferred, as others have, to wait until the deformity has stopped progressing and then recommended a plastic operation on the femoral head and neck, employing Brackett's method or the bifurcating operation of Lorenz. We agree that subtrochanteric osteotomy does not attack the site of the deformity but the deformity can be corrected by this means. By the use of this operative procedure, the femoral head may be placed in valgus, thereby changing the plane of the epiphyseal line from a vertical to an oblique line or one more nearly at right angles to the stresses of weight-bearing. This change in position will prevent recurrence of the deformity on weight-bearing and frequently will cause union of the epiphyseal disk. This union has been noted to occur in several of our patients almost as quickly as the osteotomy site has solidly united. Because of this early union of the upper femoral epiphysis following subtrochanteric osteotomy and no recurrences, we doubt the necessity of first drilling the upper femoral epiphysis as Zadek has suggested.

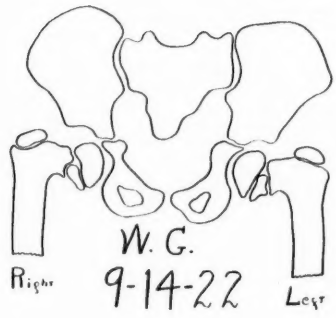
The plastic operations about the hip should be held in reserve for those patients who are seen with a residual deformity in the femoral head and neck in whom further progression of the coxa vara is impossible. In the early stages correction of the deformity should be procured by a subtrochanteric osteotomy, placing the distal fragment in sufficient abduction on the proximal so that at least the normal angle of the femoral neck with the shaft is obtained. It is a common error to undercorrect the deformity. The osteotomy is done in the usual manner, but whenever the operator has difficulty in controlling the upper fragment the insertion of a long stainless steel pin into the fragment will simplify the procedure. Also at times it may be necessary to do an adductor tenotomy.

RESULTS

Nonoperative Treatment.—In this group of 13 patients, 7 were not followed and they made only one or two visits to this hospital. Of the remaining 6 patients, subtrochanteric osteotomy was advised for 3, a shelf stabilization of the hip for 1, and nothing was advised for 2. Of these last two patients, 1 was a boy 18 years of age with marked bilateral coxa vara but with a complaint of only slight fatigue and only a slight limp. The other was a boy 14 years of age with marked coxa vara of the left hip but with six inches of shortening of this extremity.

Almost all of the patients followed continued to have their original limp and aches and pains persisted, particularly with changes in the weather, and there was no improvement in the arcs of motion of their hips.

Fig. 1.



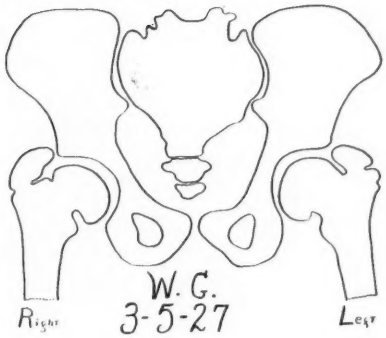


Fig. 2.

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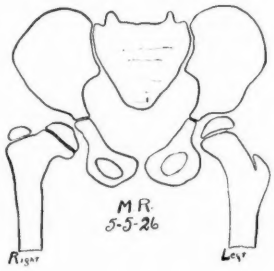

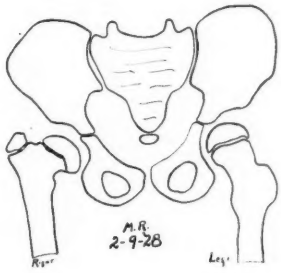

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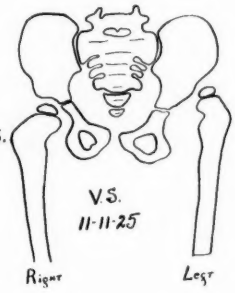

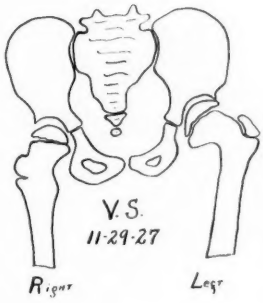

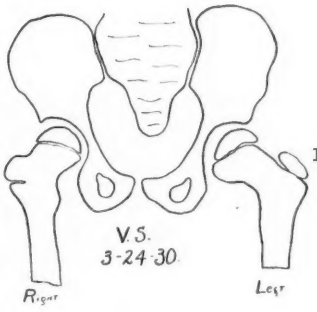

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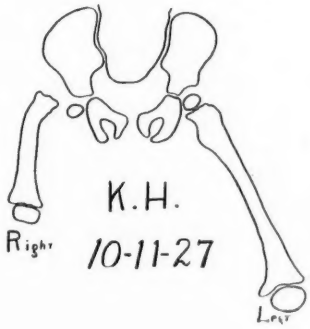

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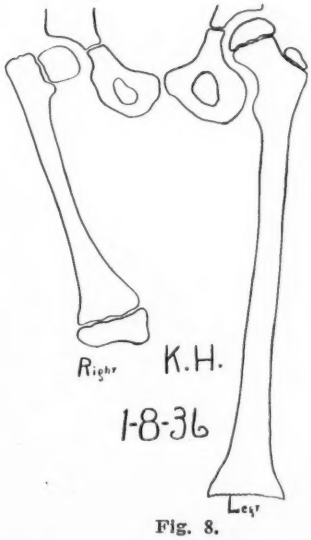

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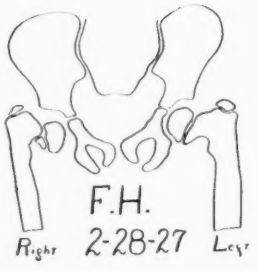

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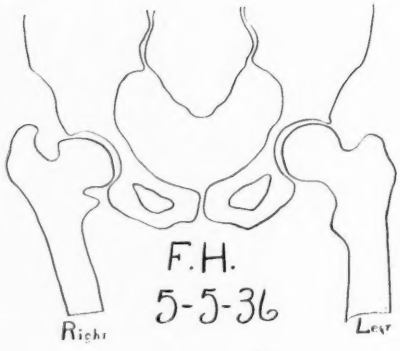

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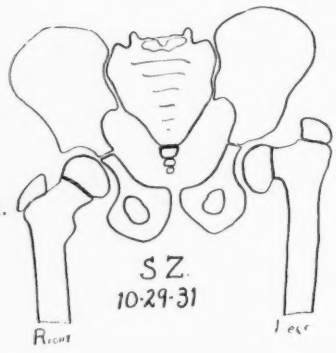

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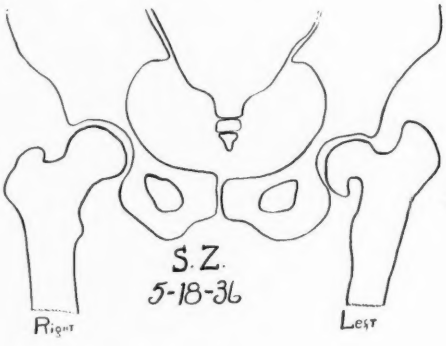

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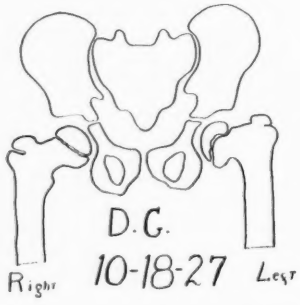

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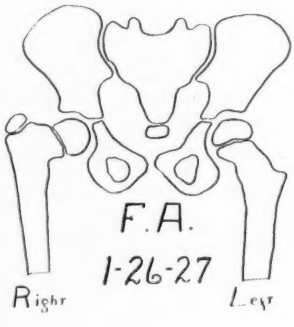

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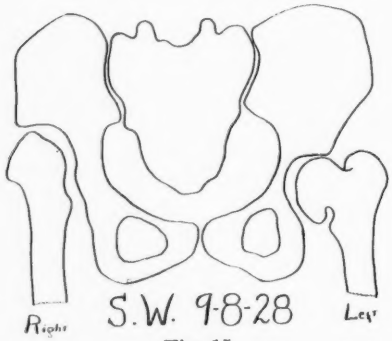

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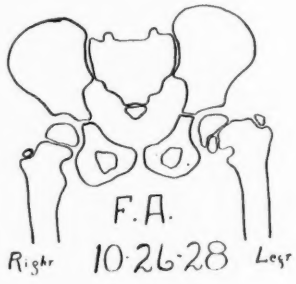

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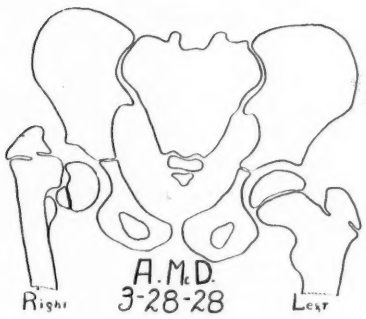

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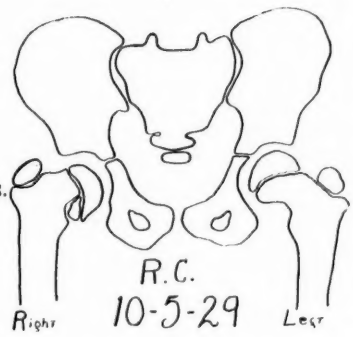

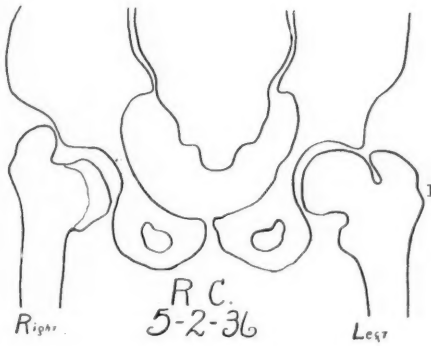

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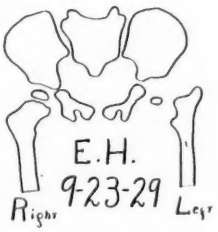

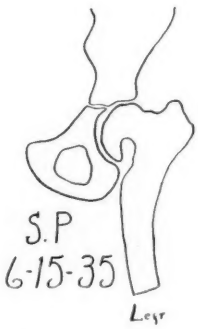

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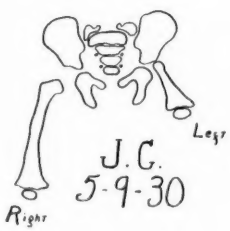

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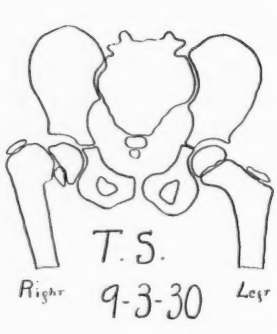

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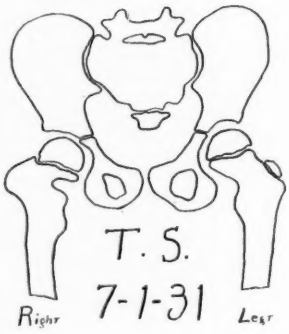

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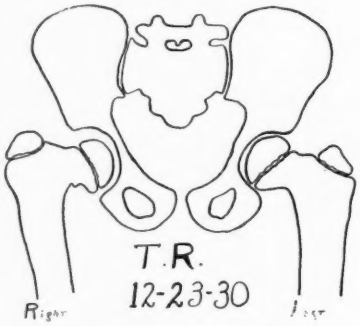

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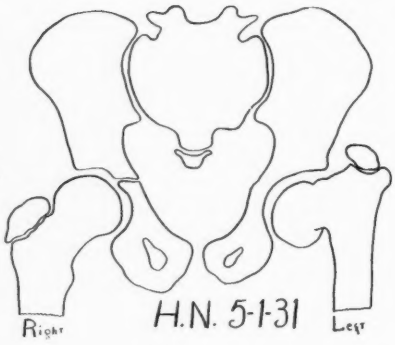

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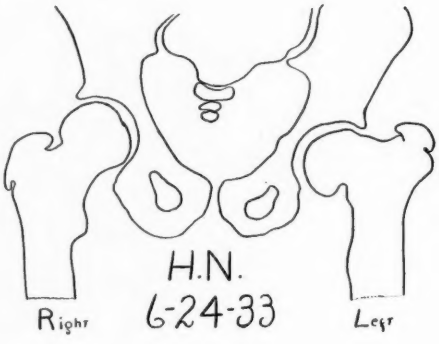

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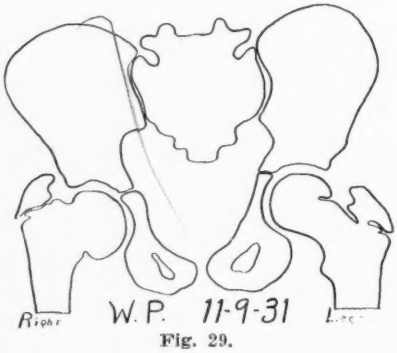

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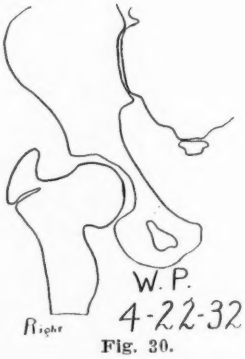

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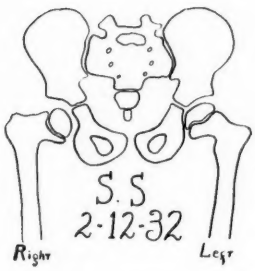

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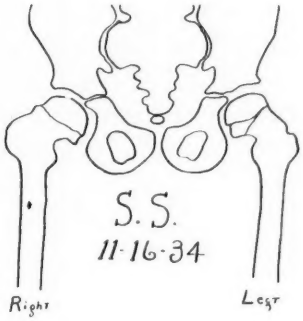

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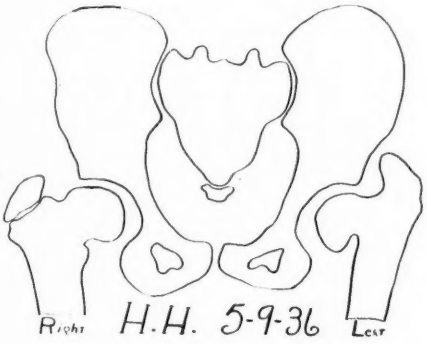

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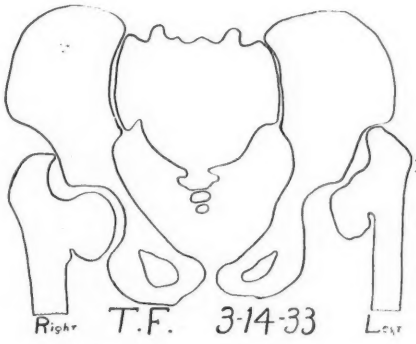

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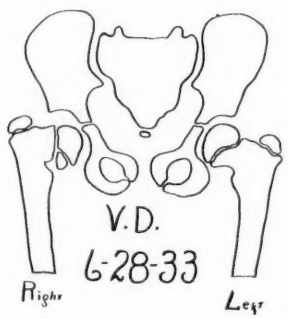


Fig. 35.

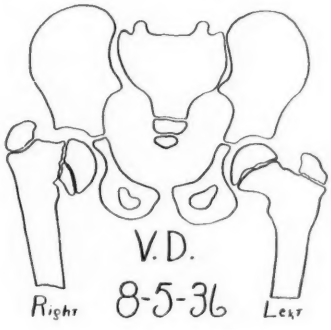


Fig. 36.

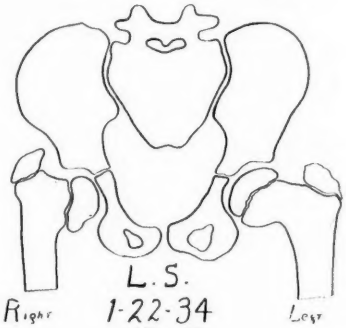


Fig. 37.

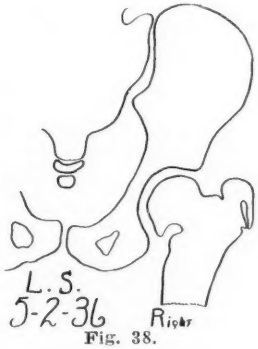
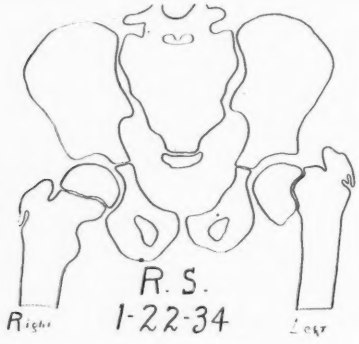


Fig. 38.

Operative Treatment.—In this group there were 18 patients, 11 males and 7 females. Coxa vara was present on the right side in 6, on the left in 6, and bilateral in 6 patients. The average age was 7.5 years, the oldest being 15 years and the youngest 15 months. There were 20 subtrochanteric osteotomies performed and 3 attempted repairs of the deformity through the upper femoral epiphysis.

Fig. 39.



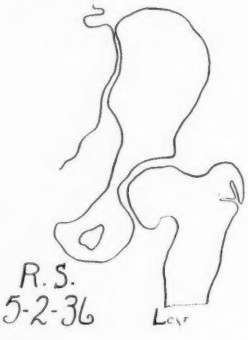
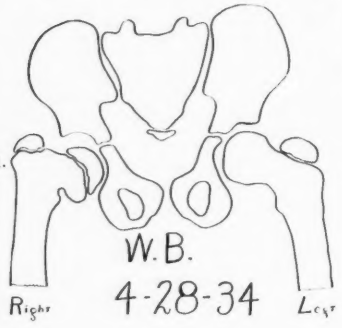


Fig. 40.

Fig. 41.



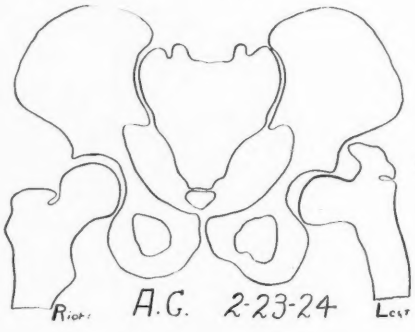


Fig. 42.

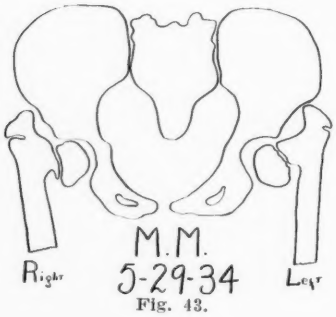


Fig. 43.

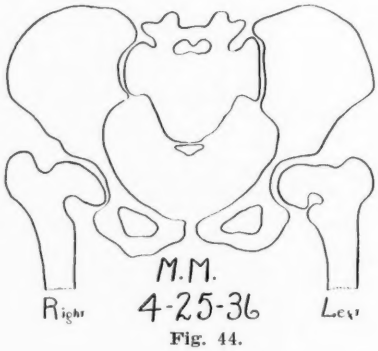


Fig. 44.

Four of the patients with bilateral deformity did not have sufficient deformity in one hip to require osteotomy.

The results in the group that had subtrochanteric osteotomy were good. There was good correction of the deformity in all patients except 2. In these 2 patients the poor result was traceable to the operator not

securing sufficient abduction at the time of operation. In a follow-up that has averaged three years, there has been no recurrence of the deformity to its previous state in any of the corrected cases. There was one factor that did progress in several of the cases and that was further shortening or absorption of the femoral neck. There were no operative fatalities, infections, or nonunions in this group. The osteotomy site

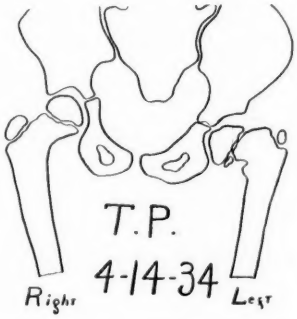


Fig. 45.

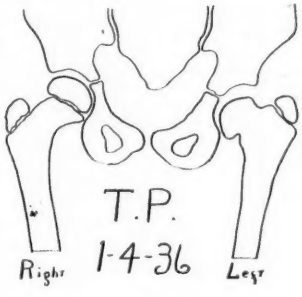


Fig. 46

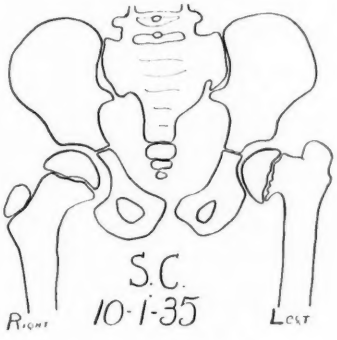


Fig. 47.

Fig. 48.

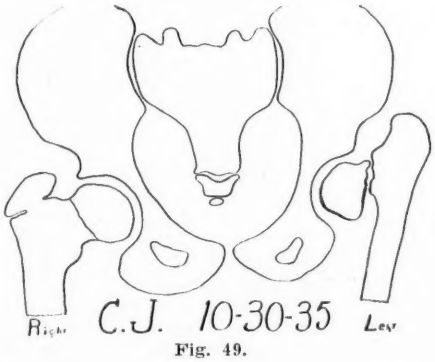


Fig. 49.

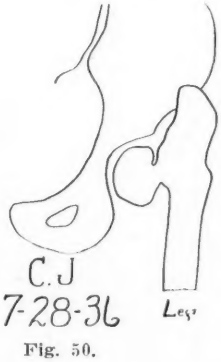


Fig. 50.

was solidly united in fourteen to sixteen weeks. Some patients no doubt would have had a much better final result than they obtained if the operator had been more courageous and had secured more abduction of the distal fragment on the proximal at the operation. The limp and feeling of fatigue have been improved in all of these patients, and the majority of them have been active in athletics since their operation.

TABLE

COXA VARA:

NAME SEX AGE HISTORY NUMBER	HIP INVOLVED DURATION OF SYMPTOMS DATE OF ADMISSION TO HOSPITAL	HISTORY AND SYMPTOMS	PHYSICAL EXAMINATION	ROENTGENOGRAPHIC EXAMINATION
1. W. G. Male 8 yr. No. 56805	Bilateral 6 yr. July 18, 1922	Has had bowed legs since starting to walk at the age of 2 years	Knees separate 2 inches on standing; greater trochanter higher than normal; abduction and internal rotation greatly reduced	Marked coxa vara at each hip; there is a supernumerary epiphysis at lower border of epiphyseal line of head in each hip
2. M. R. Female 3 yr. 69100	Right 1 yr. Nov. 16, 1923	Moderate limp has been noticed by the parents since child started walking at the age of 2 years	There was a feeling of instability and suggestion of telescoping of both hips; motion of both hips good in all directions	Moderate coxa vara right and coxa valga left; hips appear stable in internal rotation
3. V. S. Female 4 yr. 85029	Left 2½ yr. July 21, 1925	Limp and a short left leg were noted by parents when the child first started to walk; has had several attempts at closed and open reduction of a congenitally dislocated left hip made	In 1927 the left hip was well socketed but there was slight limitation of abduction and internal rotation	Left femoral head is in the acetabulum; moderate coxa vara present
4. K. H. Female 14 mo. No. 104124	Right Since birth Sept. 19, 1927	Delivery was by a version and breech extraction; short right lower extremity noted soon after birth	Right lower extremity 2½ inches shorter than the left; upper end of the femur rides well above the acetabulum	Femoral shaft appears separated from the head and displaced upward and outward about 1 inch
5. F. H. Female 5 yr. No. 78708	Bilateral 2 mo. April 20, 1927	In November, 1924, patient had rachitic genu varum; given antirachitic with improvement of the deformity; two months ago left-sided limp was noticed	Right trochanter is above Nelaton's line and the left is on a level with it; there was 15° flexion deformity of the right and 5° of left hip; abduction to 5° right and 15° left	Bilateral coxa vara with epiphyseal separation and subluxation of right femur upwards on the head; epiphyseal zones show irregularity of outline density but a smoothness of the line that suggests cessation of activity

I

CASE REPORTS

DISCUSSION CLASSIFICATION OF TYPE OF COXA VARA	TREATMENT	FOLLOW-UP; END-RESULTS	TRACINGS OF ROENTGENOGRAMS	
			PREOPERATIVE	POSTOPERATIVE
This is typical developmental type of coxa vara	Sept. 22, 1922: Subtrochanteric osteotomy, bilateral; distal fragments placed in 40° abduction	June 26, 1928: Patient walks well; no complaints; abduction free in both hips	Fig. 1	Fig. 2
This case falls into the congenital type, due to the subluxation of the femoral head in the acetabulum; treatment is to be directed toward stabilizing the hips	Nov. 28, 1923: Closed reduction of the dislocated hips July 24, 1924: Supracondylar osteotomy for anteversion	July 14, 1928: Pronounced right-sided limp; denies ache, pain, or fatigue; has ¾-inch shortening of right lower extremity; wears lift inside heel of right shoe	Fig. 3	Fig. 4
Congenital type; coxa vara was not severe	None, except for reduction of the dislocated left hip	June 12, 1930: Coxa vara gradually improved so that on this examination angle of inclination of femoral head was 115°; denies any ache, pain, or fatigue	Fig. 5	Fig. 6
This may possibly have been a birth fracture but rarity of such a lesion leaves this diagnosis open to question	Oct. 17, 1927: Hip opened; fibrous union between femoral head and side of shaft was found; this was broken up, shaft pulled down, and steel pin inserted	June 8, 1936: Patient having no symptoms, but does not walk more than a few blocks at a time; all motions of the hip are good, but has 6-inch shortening of left lower extremity, almost all of which is in femur	Fig. 7	Fig. 8
Roentgenograms and history suggest epiphyseal separation following rickets; with the cessation of activity and presentation of this deformity subtrochanteric osteotomies were advised Rachitic type (?)	Oct. 17, 1928: Subtrochanteric osteotomy, bilateral	May 5, 1936: Denies any ache, pain, or fatigue in her hips; abduction of the right hip to 20° and 40° of the left; has ½-inch shortening on the right; gait is excellent, and no limp	Fig. 9	Fig. 10

TABLE

NAME SEX AGE HISTORY NUMBER	HIP INVOLVED DURATION OF SYMPTOMS DATE OF ADMISSION TO HOSPITAL	HISTORY AND SYMPTOMS	PHYSICAL EXAMINATION	ROENTGENOGRAPHIC EXAMINATION
6. S. Z. Female 9½ yr. No. 104498	Left 3 yr. Oct. 3, 1927	When patient was 6½ years of age, she began to complain of pain in her left hip and knee; this was accompanied by some limp; no treatment during this time	Motion of the left hip was limited in all directions especially abduction; left trochanter was high; slight left-sided limp with 1-inch shortening	Marked coxa vara of the left hip; irregular structure of the neck in its middle third
7. D. G. Male 6 yr. No. 104895	Bilateral 8 mo. Oct. 18, 1927	Patient has been walking with a limp for past 8 months; denies any ache or pain; had tibial osteotomies for bowed legs two years ago elsewhere	Both hips have limited abduction to 10°; no muscle spasm or pain; has a moderate amount of genu valgum	Marked coxa vara of both hips; large fragment between left femoral head and neck; bilateral bowing of femora
8. F. A. Female 6 yr. No. 105342	Right 5 yr. Nov. 4, 1927	This child had scurvy at 6 months of age; parents have noticed that the right leg was shorter than the left and that she limps at times	No pain but muscle spasm is present at the extremes of motion; right greater trochanter is higher than the left; right lower extremity one inch short	Marked coxa vara right, with the femoral head united to the neck
9. S. W. Female 16 yr. No. 52588	Left None Sept. 8, 1928	Child was born with a congenital dislocation of the right hip and has had several operations at another hospital; no complaint about left hip but has aches and pains in right hip	Slight limitation of abduction and internal rotation of the left hip; right hip telescopes and there was a moderate range of free motion	Marked coxa vara on the left; epiphyseal line united; on the right there is no femoral head and the femur is dislocated
10. F. A. Female 6 yr. No. 89790	Left 1 yr. Oct. 10, 1928	Complains of left-sided limp; has rachitic bowed tibia for which osteoclasis was done April 19, 1926	Slight limitation of abduction; ½-inch shortening of the left lower extremity	Marked coxa vara on the left; epiphyseal line is wide and ragged; demarcated fragment present
11. A.McD. Female 10 yr. No. 108582	Right 9 yr. March 28, 1928	Has marked right-sided limp due to a short and weak lower extremity; wears a high shoe on the right foot	Right hip telescopes both upward and downward; all motions limited; has 1½ inches shortening of the right lower extremity	Femur is underdeveloped; acetabulum shows an oblique roof; femoral head has been separated from the neck; neck is short and displacement of the head downwards produces marked coxa vara

I—CONT'D

DISCUSSION CLASSIFICATION OF TYPE OF COXA VARA	TREATMENT	FOLLOW-UP; END-RESULTS	TRACINGS OF ROENTGENOGRAMS	
			PREOPERATIVE	POSTOPERATIVE
Roentgenogram is consistent with an old fracture now united; fracture may have been pathologic through a bone cyst now healed; subtrochanteric osteotomy advised to correct deformity; fracture type (?)	June 19, 1929: Subtrochanteric osteotomy, left	May 4, 1936: Patient states her gait was improved by operation and never has any ache, pain, or fatigue in her hip; abduction is to 25° on left and 35° on normal right hip	Fig. 11	Fig. 12
This is a developmental type of coxa vara; subtrochanteric osteotomies advised	None; operations refused	Nov. 22, 1928: Hips were stretched at another hospital and mother thought the child walked better; still needs subtrochanteric osteotomy to improve abduction deformity on the left	Fig. 13	No follow-up examination
This may have been an acquired coxa vara due to the history of scurvy; subtrochanteric osteotomy would improve relationship of femoral head and neck to the femoral shaft	None; subtrochanteric osteotomy refused	Not followed	Fig. 14	No follow-up
Congenital type of coxa vara; symptoms were all related to right hip; no treatment advised for left hip	Treated conservatively	April 6, 1929: Only complaint is occasional pain in first one hip and then the other; moderate amount of fatigue; all motions are fairly good	Fig. 15	No follow-up
This appears to be a developmental type but not typical; subtrochanteric osteotomy advised	Left subtrochanteric osteotomy advised	Feb. 8, 1936: Has pain in left hip during changes in the weather and any very active sports; latter pain is relieved by rest; motion good; ½-inch shortening	Fig. 16	No follow-up
Femoral head is probably united to the neck by fibrous tissue; shelf stabilization of this hip was advised; should also have a subtrochanteric osteotomy Developmental in type	None	No follow-up record	Fig. 17	No follow-up

TABLE

NAME SEX AGE HISTORY NUMBER	HIP INVOLVED DURATION OF SYMPTOMS DATE OF ADMISSION TO HOSPITAL	HISTORY AND SYMPTOMS	PHYSICAL EXAMINATION	ROENTGENOGRAPHIC EXAMINATION
12. R. C. Male 12 yr. No. 119584	Bilateral 1 yr. May 28, 1929	Complains of a right-sided limp; denies any aches or pains; only injury was a blow over the right gluteal region one year ago	Has exaggerated lumbar lordosis; abduction is limited to 10°; other motions good; has ½-inch shortening of right lower extremity	Bilateral coxa vara; marked right and slight left; epiphyseal line is wide and neck irregularly ossified on the right with a demarcated piece of bone between the femoral head and neck
13. E. H. Male 1½ yr. No. 122845	Left 1½ yr. Sept. 23, 1929	Abnormal presentation and difficult labor at childbirth; shortening of his left lower extremity was noticed soon after birth and child was quite ill with convulsions from an intracranial hemorrhage for the first year; admitted here because of his short left lower extremity	Marked telescoping of the left hip; trochanter was above Nelaton's line; mobility of the hip was good except that abduction was limited; in 1931 there was ¾-inch shortening of left lower extremity	Acetabulum shallow; roof oblique; femoral head in acetabulum; femoral neck separated from the head with ½-inch upward displacement of femur
14. S. P. Female 4 yr. No. 127477	Left 1 yr. March 29, 1930	One year ago a limp was noticed that increased in severity; onset was following a fall downstairs and at that time her leg seemed shorter; denies any ache or pain; no limitation of her activities	Left hip stable, no spasm; had 20° flexion deformity and abduction was limited to 20°; other motions good; has ½-inch shortening of left lower extremity	Marked coxa vara left hip; neck displaced upwards on head with varus angulation; end of neck irregular with some irregular union to head
15. J. G. Male 1 yr. No. 128858	Right 1 yr. May 9, 1930	Patient was born with congenital absence of the left patella and upper third of left femur; this congenital deformity was first noticed 17 days after birth of child	There was 1-inch shortening of left lower extremity on first examination; began walking at 14 months of age; Aug. 29, 1932, had 3½-inch shortening of left lower extremity	May 9, 1930: Absence of upper epiphysis of the left femur has resulted in absence of upper half of the bone; there is a coxa vara on the right, but femoral head is not ossified
16. T. S. Male 5 yr. No. 132145	Right 4 yr. Sept. 3, 1930	When child started walking at 14 mo. of age, mother noticed right leg was short and that he walked on the toes of his right foot	Right trochanter high; Trendelenburg's sign positive; abduction to 5°; internal rotation to 15°; slight limp; 1-inch shortening on right	Right femoral head displaced downwards on neck ¾ inch or more and reunited by irregular callus in marked coxa vara

TABLE I—CONT'D

DISCUSSION CLASSIFICATION OF TYPE OF COXA VARA	TREATMENT	FOLLOW-UP; END-RESULTS	TRACINGS OF ROENTGENOGRAMS	
			PREOPERATIVE	POSTOPERATIVE
Developmental type of coxa vara; subtrochanteric osteotomy advised for the right side for the limitation of motion	Subtrochanteric osteotomy right advised	May 2, 1936: States that he is not aware of any limp but that others notice it; has aches and pains in right hip during changes in weather; abduction is good but internal rotation is limited to 0° on right and 10° on left; ½-inch shortening on right	Fig. 18	Fig. 19
This is a congenital type of coxa vara; reconstruction of hip was advised at admission in 1931, with repair of the defect between the femoral head and neck	May 4, 1931: Reconstruction left hip; femoral head loosely united by cartilaginous tissue to the neck; there was only slight motion present between the head and neck; no remnant of ligmentum teres; no evidence of previous infection	Jan. 5, 1936: Patient is quite active with marked left-sided limp; all motions are slightly limited; has 1¾-inch shortening of left lower extremity; will need further operative work, such as a reconstruction operation, at a later date	Fig. 20	No change
This is possibly a residual of fracture of femoral neck close to epiphyseal line	Dec. 16, 1932: Subtrochanteric osteotomy left; distal fragment was placed in 45° abduction	June 15, 1935: Patient denies any symptoms and engages in all athletics; has only a very slight left-sided limp; abduction to only 15°; has 1-inch shortening of the left lower extremity	Fig. 21	
Child was too young to attempt any reconstruction operation on the left hip and coxa vara on the right was just apparent, but the femoral head was not yet ossified; operative work was to be deferred until later Congenital type	No treatment for coxa vara, right; had an unsuccessful reconstruction of left hip; Sept. 7, 1932: Deformity of left hip overshadows the coxa vara on the right	May 2, 1936: Abduction is limited to 10° on right and internal rotation to 0°; has marked increase in his lumbar lordosis and 4 inches of shortening on left due to congenital absence of the upper left femur; treatment has been directed only to the left hip	Fig. 22	Fig. 23
It is possible that displacement here occurred at birth, although it most likely is a developmental coxa vara	Feb. 4, 1931: Subtrochanteric osteotomy, right; distal fragment was abducted to about 55° on the neck	Feb., 1933: Occasionally has pain in his right hip; abduction is possible to 45° on the right; has only ¾-inch shortening of right lower extremity; good result	Fig. 24	Fig. 25

TABLE

NAME SEX AGE HISTORY NUMBER	HIP INVOLVED DURATION OF SYMPTOMS DATE OF ADMISSION TO HOSPITAL	HISTORY AND SYMPTOMS	PHYSICAL EXAMINATION	ROENTGENOGRAPHIC EXAMINATION
17. T. R. Male 9 yr. No. 135055	Bilateral 2 yr. Dec. 25, 1930	Earliest history is not known; limp has been known to be present by present guardians for the past 2 years; no subjective symptoms	Right trochanter high; Trendelenburg's sign positive; abduction limited to 10° and internal rotation, to 15°, had ¼-inch shortening on the right	Bilateral coxa vara, worse on the right where epiphyseal line of head is wide and irregular
18. H. N. Male 13 yr. No. 138661	Left 6 yr. April 27, 1931	In 1925 while walking in a field patient tripped but had no disability until one week later when he began to limp; treated by plaster spica elsewhere; was better until one month ago when limp increased	Trochanter 1 inch higher than on left; motions are limited in abduction to 20° and internal rotation, to 30°; slight muscle spasm; 1-inch shortening on left	Severe left coxa vara, due to old separation of head or fracture at birth or in infancy
19. W. P. Male 13 yr. No. 144615	Right 3 mo. Nov. 6, 1931	Three months ago patient began having pain in lower end of right thigh, most severe at night; no history of injury; also slight limp was present	No pain or spasm present on moving hip; only limitation of motion was internal rotation, which was 0°; 1-inch shortening on right	Neck much smaller than head and apposed to superior half of head in coxa vara
20. S. S. Male 3 yr. No. 147116	Bilateral 1½ yr. Feb. 10, 1932	At 1½ years of age pediatrician noted a waddle in the child's gait	Slight waddle in his gait; abduction was limited to 20° right and 25° left; slight spasm on abduction	Bilateral coxa vara; epiphyseal line wide, ragged in appearance
21. H. H. Male 11 yr. No. 152142	Bilateral 8 yr. July 15, 1932	Patient has had slight limp and occasional ache and pain in both hips for past 8 years; occasionally pain has been very severe	Increased lumbar lordosis; trochanters high; abduction limited to 15° right and 10° left; left lower ½ inch short	Bilateral coxa vara; extreme on the left
22. T. F. Male 18 yr. No. 158585	Bilateral 17 yr. March 14, 1933	Patient has had left-sided limp ever since he started to walk; no aches or pains	Increased lumbar lordosis; trochanters high; abduction limited to 15° on both sides; left lower ¾ inch short	Each femoral head has been displaced downward on the neck which is underdeveloped; marked coxa vara; left acetabulum shallow with roof very oblique; left femoral head irregular

TABLE I—CONT'D

DISCUSSION CLASSIFICATION OF TYPE OF COXA VARA	TREATMENT	FOLLOW-UP; END-RESULTS	TRACINGS OF ROENTGENOGRAMS	
			PREOPERATIVE	POSTOPERATIVE
Developmental type of coxa vara; subtrochanteric osteotomy advised	None, as parents objected to operative procedures	Feb. 23, 1932: No change in the arcs of motion in either hip; still has a slight limp	Fig. 26	No change
Patient advised to have a subtrochanteric osteotomy; fracture or epiphyseal separation	July 17, 1931: Subtrochanteric osteotomy, left; distal fragment was placed in only 15° abduction	June 9, 1934: No symptoms referable to hip; no pain, tenderness, or spasm; abduction to 20° and internal rotation to 10° left hip	Fig. 27	Fig. 28
Apparently this is the result of an early traumatic separation rather than adolescent slipping; subtrochanteric osteotomy on the right side advised	Nov. 27, 1931: Subtrochanteric osteotomy, right; distal fragment placed in 30° abduction	April 20, 1932: Denies any pain or fatigue; walks actively but with a slight right-sided limp; abduction to 30° and internal rotation to 5°; has 1½-inch shortening on the right	Fig. 29	Fig. 30
Bilateral subtrochanteric osteotomies advised; developmental coxa vara	Feb. 19, 1932: Subtrochanteric osteotomy, right March 21, 1932: Subtrochanteric osteotomy, left	Nov. 16, 1934: No complaints; walks with a gait that still has a slight waddle but much improved over former gait; no shortening; has a free range of motion in both hips	Fig. 31	Fig. 32
Bilateral subtrochanteric osteotomies advised; this is a developmental type of coxa vara	Nov. 32, 1932: Subtrochanteric osteotomy, left Dec. 19, 1932: Subtrochanteric osteotomy, right	May 25, 1936: No complaints; can run and walk long distances; plays all games; has ¼-inch shortening on the left; internal rotation to 10° on the right is only limitation of motion in either hip	Films lost	Fig. 33
Congenital type of coxa vara; probably present at birth or the result of a birth injury; for lack of symptoms surgery did not seem justified in this patient	None	Has not been followed up	Fig. 34	No follow-up

TABLE

NAME SEX AGE HISTORY NUMBER	HIP INVOLVED DURATION OF SYMPTOMS DATE OF ADMISSION TO HOSPITAL	HISTORY AND SYMPTOMS	PHYSICAL EXAMINATION	ROENTGENOGRAPHIC EXAMINATION
23. V. D. Female 6 yr. No. 162341	Bilateral 1 mo. June 26, 1933	For about 1 month child has been complaining of pain about right hip; limp has been noticed; complains more towards end of the day	Trendelenburg's sign was negative; has pain on extreme abduction of the right hip; abduction is limited to 10° right	Marked bilateral coxa vara; extreme on the right
24. L. S. Male 11 yr. No. 167350	Bilateral 10 yr. Dec. 28, 1933	Mother has noticed that ever since walking child has walked on his toes; complains of fatigue in feet and legs; has an identical twin	Right trochanter was high; 20° flexion deformity and abduction was limited to 10° on the right; right lower ¾ inch short	Bilateral coxa vara, very marked at the right
25. R. S. Male 11 yr. No. 367351	Bilateral 10 yr. Dec. 28, 1933	History the same as Case 24; he is the identical twin of the above patient	Left trochanter high; abduction is limited to 20° and internal rotation to 20° in the left hip	Bilateral coxa vara, marked on the left
26. W. B. Male 9 yr. No. 170998	Bilateral 9 yr. April 28, 1934	Right leg has been shorter since birth; has pains at times in right knee	Right trochanter high; increased lumbar lordosis; abduction is limited to 20° and internal rotation to 25° on the right; right lower extremity 1 inch short	Bilateral coxa vara; marked on the right; femora bowed
27. A. G. Male 14 yr. No. 168834	Left 14 yr. Feb. 23, 1934	Patient was born with a deformity of his left leg; only complaint is shortening of the left lower and occasional fatigue of the left hip	Left trochanter high; there is 10° flexion deformity; hip is held in 45° external rotation and has only 35° abduction on the left; left lower 6 inches short	Marked left coxa vara with femoral head and neck small and femur bowed laterally in upper half; bones at left knee slightly underdeveloped
28. M. M. Female 11 yr. No. 172127	Bilateral 6 yr. May 28, 1934	Patient did not walk until 3 years old; parents did not notice swaying gait she now has until she was 5 years of age; has early fatigue in her hips	Increased lumbar lordosis; all motions of her hips were reduced; no abduction in either hip; trochanters are high	Bilateral coxa vara; very severe
29. T. P. Female 6 yr. No. 170447	Right 4 mo. April 14, 1934	Occasional right-sided limp for the past 4 months; denies any ache, pain, or fatigue	Trochanter is high; abduction is limited to 10° and internal rotation to 5° on the right	Right coxa vara with neck short and its epiphyseal line irregular

TABLE I—CONT'D

DISCUSSION CLASSIFICATION OF TYPE OF COXA VARA	TREATMENT	FOLLOW-UP; END-RESULTS	TRACINGS OF ROENTGENOGRAMS	
			PREOPERATIVE	POSTOPERATIVE
Developmental coxa vara; subtrochanteric osteotomy advised	Aug. 12, 1936: Subtrochanteric osteotomy	Too early for record follow-up	Fig. 35	Fig. 36
This is a developmental type; right subtrochanteric osteotomy advised	Jan. 22, 1934: Subtrochanteric osteotomy, right; distal fragment placed in 25° abduction	May 2, 1936: Has no symptoms, plays all games, and recently walked forty city blocks; abduction is only to 20° on the right and has 1 inch of shortening on the right lower	Fig. 37	Fig. 38
This is a developmental type of coxa vara; left subtrochanteric osteotomy advised	Jan. 22, 1934: Subtrochanteric osteotomy, left; distal fragment placed in 30° abduction	May 2, 1936: Patient denies any ache, pain, or fatigue; he runs and plays all games without any handicap; abduction to 20° left; has ¾ inch of shortening of the left lower	Fig. 39	Fig. 40
Developmental type; subtrochanteric osteotomy advised	None; father refused operation that was advised	Patient was not followed	Fig. 41	No follow-up
Coxa vara on the left appears more like a residual of rickets, but only a congenital deformity would explain marked shortening; leg equalization operation or amputation was only operative procedure contemplated	None	To consider leg equalization operations for this patient	Fig. 42	No change
This is a fetal or birth injury type; subtrochanteric osteotomies advised	July 11, 1934: Subtrochanteric osteotomy, right Oct. 24, 1934: Subtrochanteric osteotomy, left	April 25, 1936: No complaints; believes her gait has been improved a great deal; abduction is to 25° in both hips; has ½-inch shortening of left lower extremity; slight left-sided limp	Fig. 43	Fig. 44
Developmental type of coxa vara; right subtrochanteric osteotomy advised	June 11, 1934: Subtrochanteric osteotomy, right; distal fragment placed in 30° abduction	April 25, 1936: No complaints; she is now a tap dancer; has no limp; abduction is to 20° on the right; has 1-inch shortening on the right	Fig. 45	Fig. 46

TABLE

NAME SEX AGE HISTORY NUMBER	HIP INVOLVED DURATION OF SYMPTOMS DATE OF ADMISSION TO HOSPITAL	HISTORY AND SYMPTOMS	PHYSICAL EXAMINATION	ROENTGENOGRAPHIC EXAMINATION
30. S. C. Male 9 yr. No. 186683	Left Unknown Oct. 5, 1935	Brought her because of left-sided limp, duration of which was unknown by father; denies aches or pain	Left trochanter high; there was limitation of abduction to 15° and internal rotation to 0° on the left; also the left lower was 1 inch short	Marked left coxa vara, with wide irregular epiphyseal line
31. C. J. Male 15 yr. No. 187487	Bilateral 7 yr. Oct. 30, 1935	Onset of pain in left hip eight years ago without history of trauma; has not had any treatment but has noticed his left leg becoming shorter	Increased lumbar lordosis; motions of right hip are good; in the left abduction is 0° and internal rotation 20°; has 1½ inch of shortening of the left lower	Bilateral coxa vara, with extreme deformity on the left; lesser trochanter is at level of middle of femoral head

There were 3 patients upon whom a repair of the deformity at the epiphyseal line was attempted. Two of the patients were babies, 18 and 22 months of age respectively, and the third was a boy of 15 years. There was no improvement in the deformity in either of these 3 cases and no doubt the epiphyseal disk was injured in the two babies. The boy of 15 years of age has had such progression of his deformity since his operation that a plastic operation will probably be needed for his hip. Therefore, any procedure that may destroy or injure the epiphyseal disk must be condemned.

The deformity progresses with the growing child but it is usually not severe at the age of 6 or 8 years. Therefore, we believe it is ideal to postpone any operative procedures until this age. We prefer a subtrochanteric osteotomy as it is possible by this means to obtain and maintain correction of the coxa vara deformity.

The accompanying table gives a summary of the 31 cases of coxa vara.

SUMMARY

Thirty-one cases of congenital and developmental coxa vara are presented. The various etiological theories are presented and the most logical one is an embryonic vascular disturbance that upsets the ossification of the femoral neck.

The signs of coxa vara predominate over its symptoms. It is a deformity that tends to progression with weight-bearing. Therefore, as soon as the diagnosis is made, an effort should be made to restore the

I—CONT'D

DISCUSSION CLASSIFICATION OF TYPE OF COXA VARA	TREATMENT	FOLLOW-UP; END-RESULTS	TRACINGS OF ROENTGENOGRAMS	
			PREOPERATIVE	POSTOPERATIVE
Developmental type; left subtrochanteric osteotomy advised	Feb. 3, 1936: Subtrochanteric osteotomy, left; long nail was employed to control proximal fragment	June 23, 1936: No symptoms; abduction is to 35°; has a moderate amount of genu valgum present	Fig. 47	Fig. 48
Developmental type; advised subtrochanteric osteotomy; at first they decided to do a repair of defect in the femoral neck	April 3, 1936: Open repair fusion defect neck of left femur with insertion of nail June 29, 1936: Nail removed	Aug. 4, 1936: No bony union present at repair site; deformity has increased; will probably need a plastic operation of the left hip now; believe a subtrochanteric osteotomy would have improved his hip previously	Fig. 49	Fig. 50

normal angulation of the femoral head and neck with the shaft of the femur. This is best done by a subtrochanteric osteotomy. This we believe is the conservative form of treatment in spite of a surgical procedure.

REFERENCES

1. Fairbanks, H. A. T.: Infantile or Cervical Coxa Vara, The Robert Jones Birthday Volume. A Collection of Surgical Essays, New York, 1928, Oxford University Press, p. 225.
2. Sutro, C. J.: Slipping of the Capital Epiphysis of the Femur in Adolescence, Arch. Surg. **31**: 356, 1935.
3. Hoffa, A.: Congenital Coxa Vara, Deutsche med. Wchnschr. **31**: 1257, 1905.
4. Jones, Robert: Quoted by Elmslie, Lancet **1**: 413, 1907.
5. Elmslie, R. C.: Coxa Vara, Its Pathology and Treatment, London, 1913, Henry Frowde.
6. Delitala, F.: Coxa Vara Congenita, Arch. di orthop. **30**: 382, 1913.
7. Nilsonne, H.: Contribution to the Knowledge of Congenitay Coxa Vara, Acta. radiol. **3**: 383, 1924.
8. Camitz: In discussion of Nilsonne's paper, Acta. radiol. **3**: 383, 1924.
9. Barr, J. S.: Congenital Coxa Vara, Arch. Surg. **18**: 1909, 1929.
10. Zadek, I.: Congenital Coxa Vara, Arch. Surg. **30**: 62, 1935.
11. Alsberg, A.: Theory and Differential Diagnosis of Congenital Coxa Vara, Ztschr. f. orthop. Chir. **7**: 365, 1899.
12. Kredel, L.: Coxa Vara Congenita, Zentralbl. f. chir. **23**: 969, 1896.
13. Reiner, M.: Relation of Congenital Coxa Vara to Congenital Defect of Femur, Ztschr. f. orthop. Chir. **12**: 297, 1903.
14. Bohm, M.: Quoted by Nilsonne and Fairbanks.
15. Helbring, C.: Coxa Vara: An Analysis of the Material From Hoffa's Private Clinic, Ztschr. f. orthop. Chir. **15**: 502, 1906.
16. Noble, T. P., and Hauser, E. D. W.: Coxa Vara, Arch. Surg. **12**: 501, 1926.
17. Duncan, George A.: Congenital Coxa Vara Occurring in Identical Twins, Am. J. Surg. **37**: 112-115, 1937.

CANCER OF THE BREAST IN THE MALE

With a Report of Four Cases and Illustrations of a Simplified Radical Mastectomy

Jacob Sarnoff, M.D., Brooklyn, N. Y.
(From the Israel Zion Hospital)

FOR every cancer of the breast in the male there are 100 carcinomas of the breast in the female. We can conclude, therefore, that it is a rather rare occurrence in the male. Although it is more readily recognized and is diagnosed earlier in the male because of the absence of surrounding fat and glandular tissue, yet carcinoma of the breast in the male is more apt to recur and has a higher mortality rate. The reason may be that there is less of a lymphatic barrier in the male, as the distance is much shorter between the nipple and the chest wall and the viscera as well.

In a recent lecture Dr. John F. Erdman stated that during his surgical career (which we know has been most extensive) he operated on five cases of carcinoma of the breast in the male. My curiosity was aroused, and I wondered how many cases of carcinoma of the breast in the male I could trace in my experience. Four cases came to my mind. Two of these I remember having operated upon within the last year. Recently I saw these patients in my routine follow-up. The other two cases that came to my mind were operated upon three and ten years ago.

I was doubtful as to whether they were still alive; however, I traced them both and found that they were in good health. I was anxious to photograph and record these four cases as a group. To this they gladly consented. Their psychology was most interesting. No matter how tactful the physician or surgeon is in masking the identity of the lesion of cancer of the breast to the patient, they somehow subconsciously feel what they really have had, although they do not admit it. This was well illustrated in the conversation that ensued between them. They acted like buddies who were in the trenches and who had escaped the ravages of the enemy's attack. They narrated and compared their experiences. The last two patients, operated upon one year and six months ago, heaved a sigh of relief when they saw the other two men, operated upon three and ten years ago, in the best of health. It gave them a new lease on life.

The diagnosis of the cases operated upon by me at the Israel Zion Hospital was corroborated by the gross and microscopic pathologic

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reports. In the last three cases the preoperative findings, the retraction of the nipple with a hard lump, as well as the operative technique, dissection of the pathologic specimens, and the end-results, have been recorded by the cinema.

CASE REPORTS

CASE 1.—B. D. a man aged fifty-three years, was operated upon ten years ago for carcinoma of the right nipple. In this case there was no enlargement of the axillary lymph glands. Operation: The nipple and a good segment of the sur-

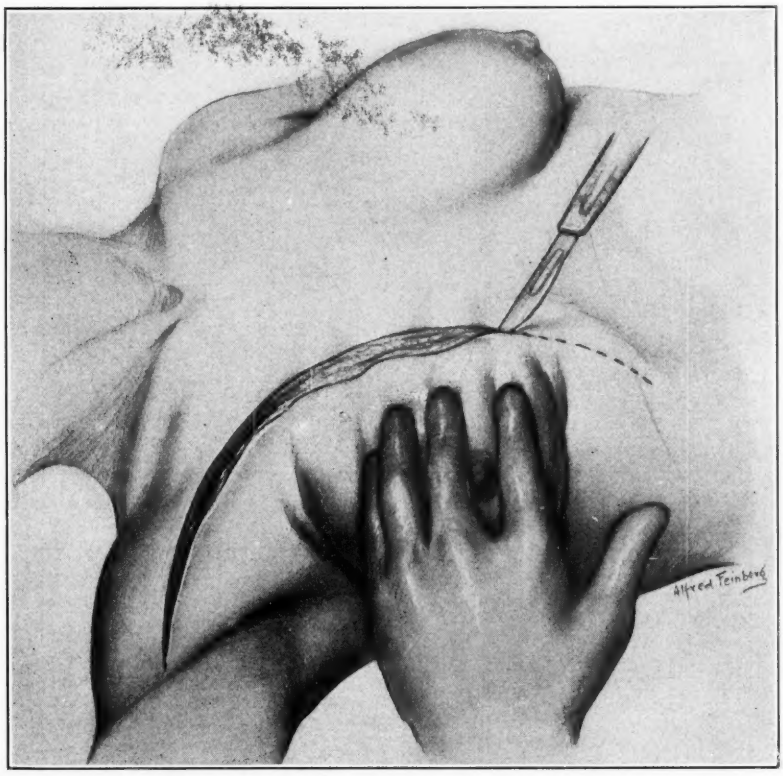

Fig. 1.—Simplified radical mastectomy; inner half of the elliptical incision is made through the skin and fascia while the breast is retracted laterally.

rounding skin, deep and superficial fascia, and pectoral fascia were excised "en masse" by an elliptical incision in the transverse direction about five inches long and three inches wide. The wound was sutured and healed by primary union. Recovery was uneventful and now, ten years later, the patient is in good health.

CASE 2.—J. C., a man aged fifty-one years, was operated upon three years ago. He had noticed a hard mass of the right nipple for ten months. Physical examination revealed a mass the size of a hazelnut involving the right nipple. It was hard, not tender, no axillary glands were palpable. Diagnosis: Carcinoma of right nipple. Operation: The nipple with a large area of the surrounding skin, fascia, and the underlying pectoralis major muscle together with the axillary glands was excised

by an elliptical incision in a vertical direction. Wound healed by primary union. Pathologic Report: Solid and tubular carcinoma of male breast. Recovery in this case was uneventful.

CASE 3.—D. P., a man aged fifty-three years, noticed a lump in the left breast four months before. Shortly after, he noticed some lumps in the axilla. Physical

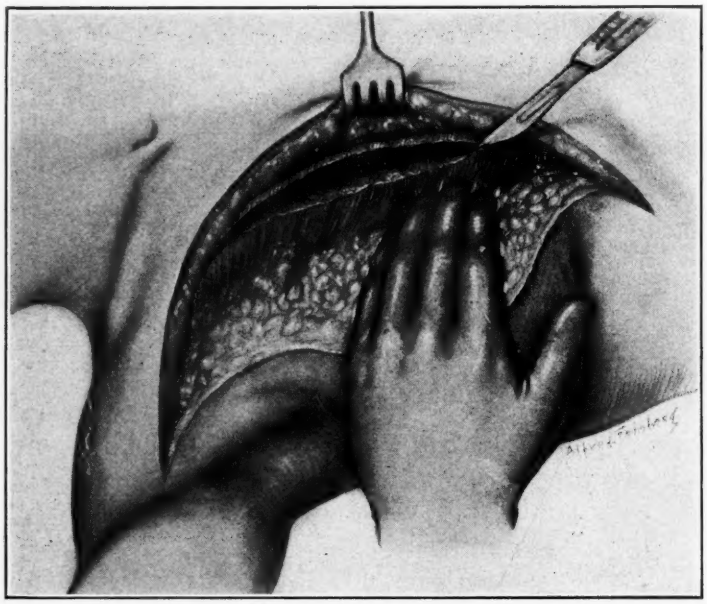

Fig. 2.—The costal and clavicular origin of the pectoralis major is then cut across.

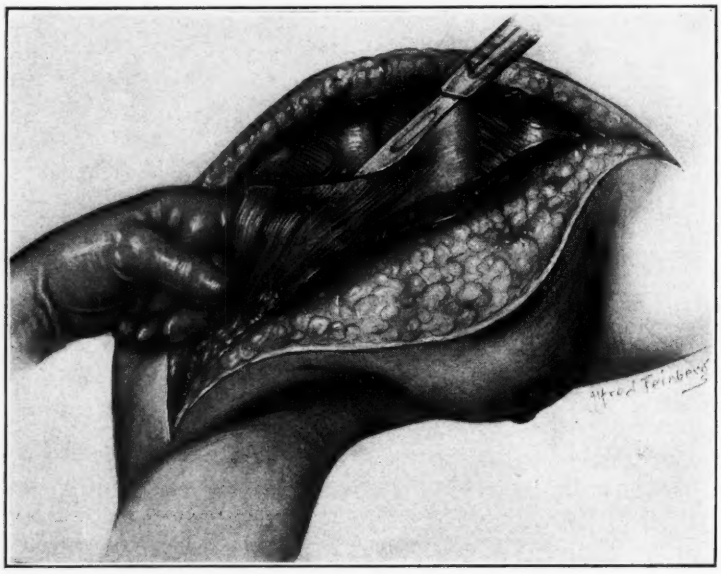

Fig. 3.—The pectoralis minor is cut away from its origin of the third, fourth, and fifth ribs.

Examination: The mass around the nipple was the size of a walnut, not tender; the nipple was retracted, glands were palpable in the left axilla. Diagnosis: Carcinoma of left nipple with metastasis to axillary glands. Operation: In this case a "simplified radical mastectomy" was performed, removing the growth together with the pectoralis major and minor muscles and the axillary glands. Pathological Report: Scirrhous tubular carcinoma with metastasis to axillary lymph glands. Recovery was uneventful. The patient was given a series of X-ray treatments postoperatively.

CASE 4.—S. P., a man aged forty-eight years, a presser, was operated upon six months ago. He noticed a lump in the right breast seven months previously. Physical examination showed retraction of right nipple with a mass about the size of a walnut with extensive metastasis of axillary glands. Diagnosis: Carcinoma of right breast with metastasis. A "simplified radical mastectomy" was performed.

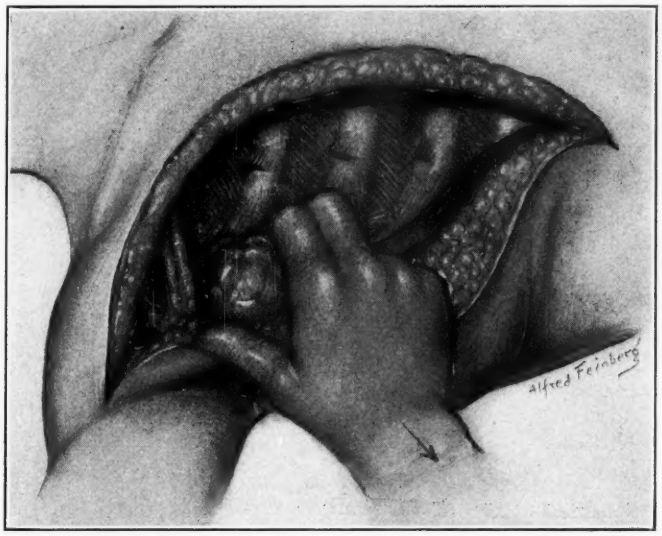

Fig. 4.—After the pectoral muscles are severed from their origin and insertion, exposing the axillary contents, the metastatic glands and axillary fat are dissected away from the axillary vessels.

Wound healed by primary union. Patient received postoperative X-ray therapy for a number of months. During this time he had quite some edema of the right arm and forearm. This, no doubt, was due to the lymphatic and possibly venous stasis, following the extensive resection of the involved glands of the axillary space and around the axillary vessels.

It may be of interest at this point to explain what is meant by the author's "simplified radical mastectomy," as compared to the ordinary radical mastectomy operation for carcinoma of the breast. The operation of radical mastectomy for carcinoma of the breast has many labels attached to the procedure: the Halsted, Willy Meyer, Kocher, etc. Each one claims some originality of advantage, yet all remind one of removing a tree by chopping off limb by limb and cutting across

the trunk and then removing its roots and all. By cutting the trunk first, one would obviate unnecessary pruning of the branches, so too by the simplified radical mastectomy, the operative steps are considerably shortened.

In all accepted procedures we first make an elliptical incision around the breast, in one form or another; cut the skin all around, then the fascia, then the muscles on either side, where we encounter a great many blood vessels—and still more blood vessels until we reach the axillary space. We separate the various glands from surrounding structures and then remove the breast. During this process of cutting

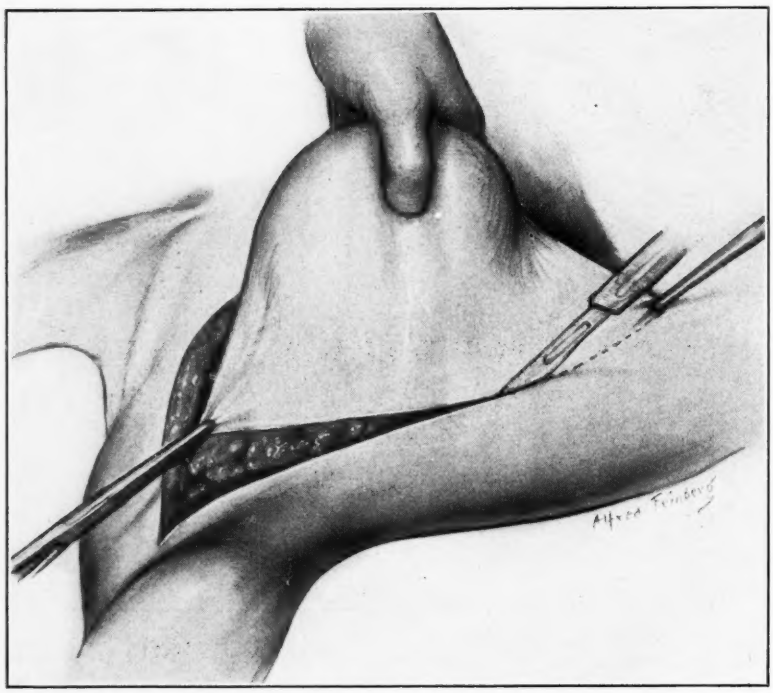

Fig. 5.—The outer half of the elliptical incision is then made by one stroke of the knife, held obliquely to remove more of the fascia than skin.

we are apt to encounter malignant breast tissue and metastatic glands in the effort to complete the radical mastectomy.

To obviate the aforementioned technical and pathologic handicaps, the writer has simplified a radical mastectomy in the following manner. This procedure is schematically presented in the accompanying illustration.

The operation for radical mastectomy is begun by first making only the inner half of the elliptical incision through the skin, extending from the coracoid process to the costal margin (Fig. 1). The skin is then undermined and enough of the fascia is thus exposed and re-

moved together with the costal and clavicular origin of the pectoralis major by a few bold strokes of the scalpel from above down, while the pectoral muscle and fascia are held on tension outwards with the palm of the other hand (Fig. 2).

As the origin of this muscle is cut across and retracted laterally, the anterior chest walls with the intercostal muscles and ribs are exposed to view, together with the origin of the pectoralis minor, which

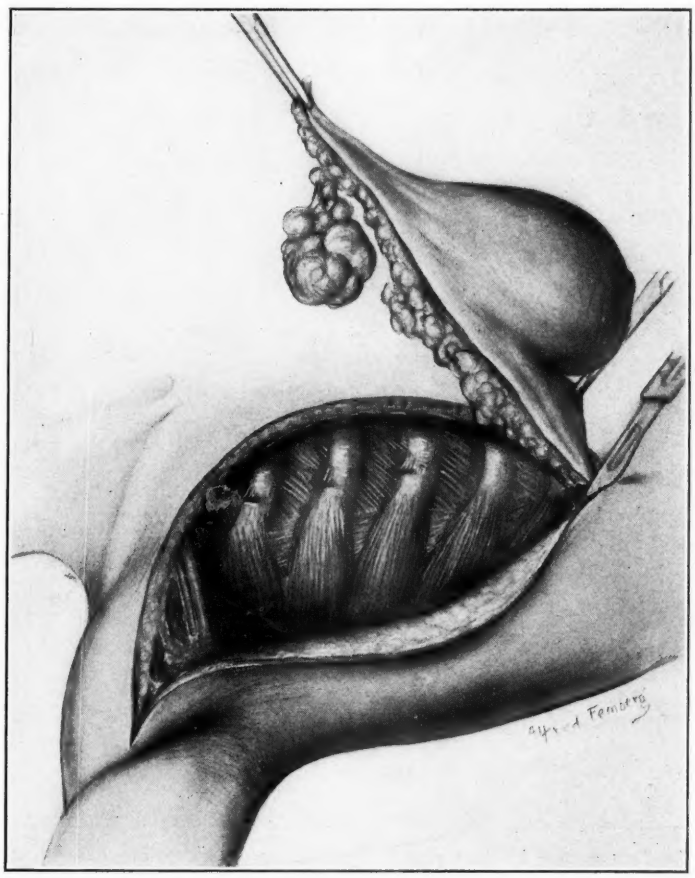

Fig. 6.—The breast is now readily removed "en masse," leaving the axillary space cleared of all metastatic glands.

is likewise severed. (Fig. 3.) Thus, with a few strokes of the knife and with a severance of only a few blood vessels, the axillary space is laid wide open and exposed. The long thoracic nerve, the serratus magnus and subscapular muscles, and the free border of the teres major and latissimus dorsi, as well as the brachial plexus and vessels, are in clear view.

By inserting the tips of a few fingers into the apex of the axilla and with the aid of a flat sponge, the axillary metastatic glands and

the surrounding fat are milked away from the brachial plexus, the axillary vessels, and the axillary wall (Fig. 4). All that remains now to be done is to ligate a few small vessels, cut across the insertion of the pectoralis major away from along the outer bicipital groove and the pectoralis minor from the coracoid process, leaving the breast with the metastatic glands attached merely by the outer aspect of the skin.

The breast is now held up taut by an assistant and a lap sponge placed in the axilla to protect the chest wall and with one sweep the outer half of the elliptical skin incision is completed, thus removing

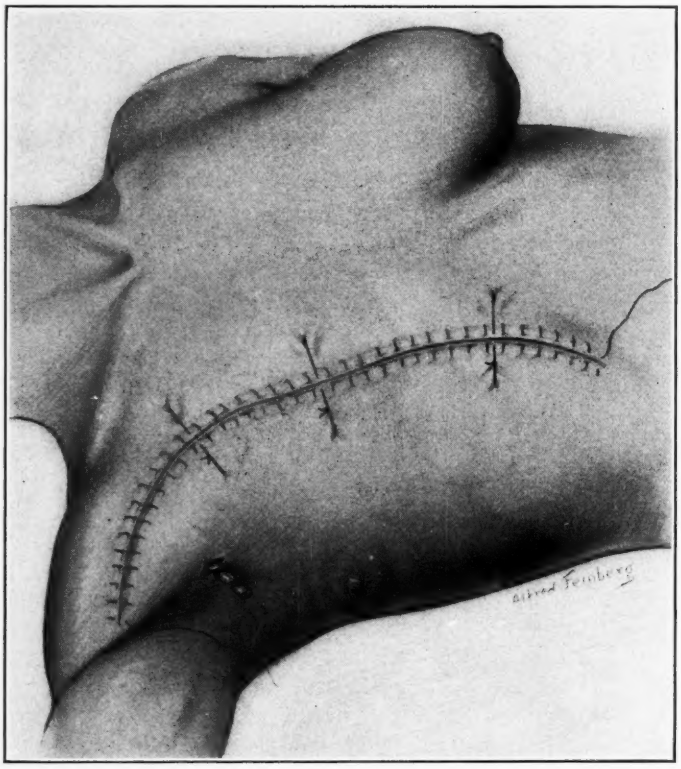

Fig. 7.—The wound is then readily sutured using kaldermic figure-of-8 tension sutures and the author's superficial and deep continuous silk for the skin edges. A small spiral drain is introduced through a puncture at the apex of the axilla.

the breast, pectoral muscles, and axillary contents "en masse" (Fig. 5) with the least severance of blood vessels and handling of malignant tissue and with a minimum of bleeding and use of ligatures (Fig. 6).

By this procedure, one also is in a better position to decide how much skin is to be removed, as one can judge the approximation with more certainty after such mobilization (Fig. 7). The length of the operation by this method can be cut in half without the sacrifice of thoroughness or loss of tissue. The procedure, in short, is a radical

mastectomy, but the inner elliptical skin incision is made first and the outer incision is completed only after all the structures have been dealt with. The closure of the skin and drainage are the same as in the other methods of radical mastectomy.

CONCLUSION

A report is presented of four cases of carcinoma of the breast in the male. The follow-up shows the recovery of the four consecutive cases of carcinoma of the breast in the male operated on six months, one year, three years, and ten years ago. A method is described to simplify the procedure of radical mastectomy which lessens the handling of tissues, facilitates the control of bleeding, and minimizes the length of the operation without the sacrifice of thoroughness.

A MODEL TO ILLUSTRATE THE MECHANICS OF RESPIRATION AND MOVEMENTS OF THE MEDIASTINUM WITH AND WITHOUT VARIOUS KINDS AND DEGREES OF BRONCHIAL STENOSIS

L. Eloesser, M.D., and Anthony Freeman, San Francisco, Calif.

(From the Department of Surgery, Stanford University Medical School at the San Francisco Hospital)

A MODEL to illustrate the mechanics of respiration, the movements of the mediastinum, and the effects of impairing respiratory movement of the lung awakened interest at the last meeting of the American Association for Thoracic Surgery and at other meetings when it was shown. Several requests were received for specifications. Since the model may be constructed by any good mechanic, the specifications are given below.

The apparatus consists of a celluloid or cellulose acetate thorax, bound in metal, mounted on a base, and divided into equal halves by a rubber mediastinum. Each half is entered by an airtight rubber cork, pierced by a glass tube, representing a major bronchus. To each cork is fastened one of a pair of well-matched toy balloons, representing a lung. Each half of the thorax, moreover, is connected to the cylinder of a valveless air pump, having two cylinders and pistons of equal bore and stroke operated simultaneously by a single handle. The upstroke of the pump exhausts air from the thorax and simulates the sucking effect of inspiration; the downstroke returns the exhausted air to the chest and simulates expiration. Thus, by moving the pump handle up and down, the balloons representing the lungs are caused to inflate and deflate. If one balloon is less easily distensible than the other, as a lung would be if it were fibrous, the mediastinum moves toward the less distensible side on inspiration.

If inflation and deflation of one balloon are uniformly impaired, as they would be in an unyielding stenosis of a major bronchus, the same deviation of the mediastinum toward the affected side occurs; moreover, the stenotic balloon with each succeeding stroke of the pump handle gradually entraps air, remaining more inflated and not emptying as completely as its unchecked neighbor. This uniform unyielding bronchial stenosis is easily simulated by inserting an ordinary hexagonal lead pencil into the round lumen of one of the glass tubes entering the corks. A check valve stenosis, in Jackson's sense, one which impairs the intake of air but which leaves its outlet unaffected, is pro-

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duced by tying a flap of sheet rubber over the outer end of one of the glass tubes. When suction is made on the pump, the rubber is sucked over the end of the tube and the corresponding balloon remains empty while its neighbor is inflated. Again the mediastinum deviates toward the checked side.

A stop valve stenosis, the reverse of the above, is simulated by tying the rubber flap over the inner end of one of the tubes, over the end which is hidden inside the balloon. Upon working the pump, air enters the corresponding balloon but cannot escape. With each succeeding stroke the balloon becomes more and more distended and this time the mediastinum deviates more and more *away from* the affected

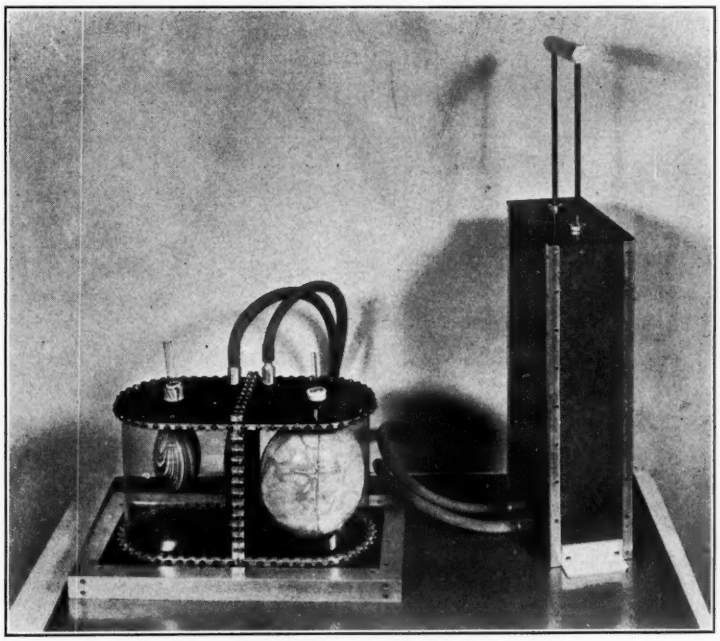


Fig. 1.—Mechanical model of thorax.

side. After three or four strokes, the air begins slowly to escape from the balloon with a funny squeak suggestive of an asthmatic wheeze.

The pump should be worked slowly, with a quicker, more forcible upstroke to simulate active inspiration and a slower, easier downstroke to simulate passive expiration.

Stopcocks have been added to the artificial pleural cavity, which provide a release. If these are not used, the rubber tubes leading from the pump to the artificial thorax must be disconnected after half a dozen strokes, for some air leaks around the pump washers and makes a release necessary.

No attempt has been made to add an artificial diaphragm at the bottom of the model thorax, as this would greatly complicate the mechanism

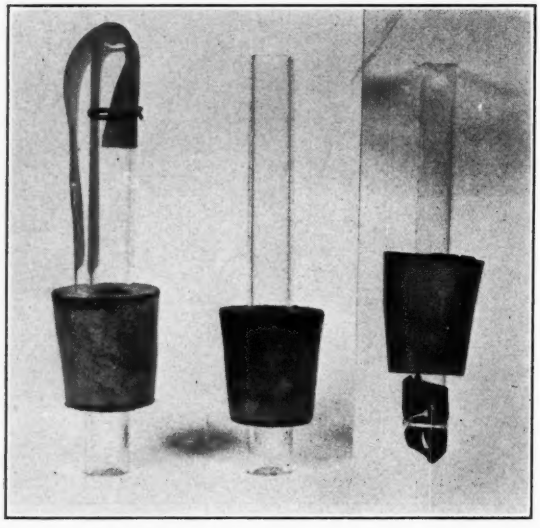
and subtract from, rather than add to, the clearness of the demonstration it affords. The suction effect of inspiration is perfectly well attained by the pump alone.

The model illustrates the mechanical effects of respiratory movements and the complications induced by the various forms of bronchial stenosis so clearly that it amply repays the trouble and work involved in its manufacture.

SPECIFICATIONS

Two chambers U-shaped in plan view and rectangular in all other elevations. The internal dimensions of the chambers are as follows:

(PLAN) Greatest length, 6 inches.
(PLAN) Greatest width, 5 inches.
Height, 7 inches.



A. *B.*

Fig. 2.—Valves to illustrate: *A*, bronchial stenosis with impaired intake (check valve); rubber flap tied over outer end of tube; *B*, bronchial stenosis with impaired outlet (stop valve); flap tied over inner end of tube.

(PLAN) The round portion of the U is a semicircle of 2½ inches radius. The straight portions are tangent to the semicircle, parallel and 3½ inches long. The external dimensions depend entirely upon the choice of materials.

The two chambers should be identical in all respects. The vertical walls are best made of celluloid 1/16 inch thick, bent roughly to a U-shape under hot water. If cellulose acetate be available, it is to be preferred as it is noninflammable. The tops and bottoms may be of bakelite, oil-tempered presswood, or any material that is sufficiently

rigid in 1/8 inch thickness. The tops should be formed to the U-shape of the chamber. The bottoms may be rectangular.

All edges are bounded with a framework of square aluminum bar bent to shape on a caul. The joints must be airtight. The framework is pierced (after bending) at intervals of about 1/2 inch with horizontal and vertical holes, capable of passing machine screws of 4-36 thread (Filaster head preferred). The holes must be staggered to avoid meeting and there should be, in all, four to the inch. The best material for the framework is pure aluminum 1/4 inch square in cross section. A lining strip of metal 1/4 inch by 1/16 inch should be provided wherever the celluloid is bound to the frame.

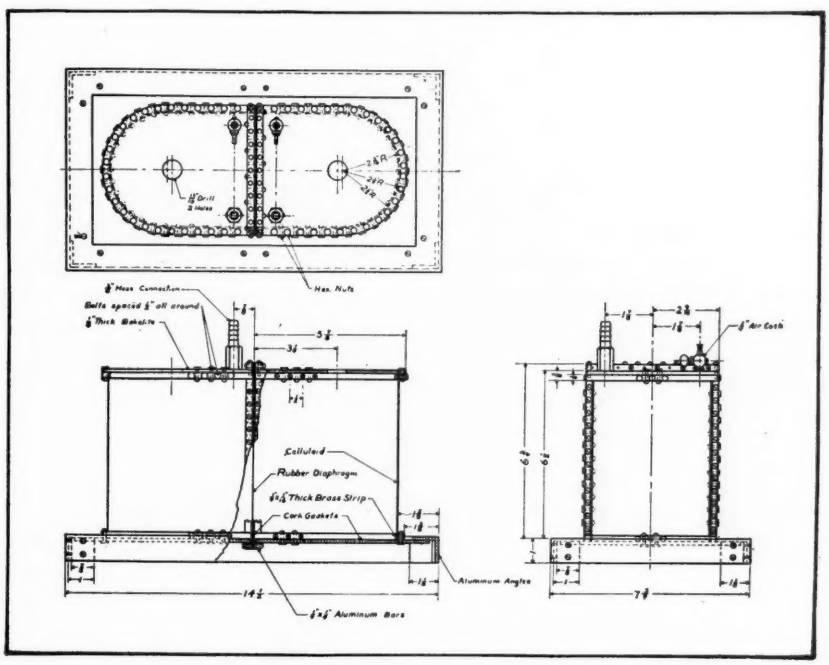


Fig. 3.—Mechanical drawing to scale.

Since the open portions of the chambers are to be fastened together, making an airtight assembly, care must be observed in drilling the holes for the screws that so connect them. If the parts are sufficiently well fitted, a light coat of shellac will serve to render them airtight.

A rubber diaphragm (light dental dam) is to be placed between the chambers. Since rubber deteriorates rapidly in contact with metal, thin gaskets of sheet cork, or better still Armstrong cork cloth, should be placed on either side of the diaphragm. The gaskets should extend inwardly 1/4 inch all around past the framework to prevent the rubber touching the ends of the machine screws. A nipple for 3/8 inch hose is to be fitted in the top of each chamber.

The bottoms of the chambers may be fitted with a frame of aluminum angle stock or feet may be provided at the corners. A hole to admit a No. 2 rubber cork is to be cut in the top of each chamber at the center of the circular portion. The cork is pierced with a hole into which is fitted a piece of ¼ inch glass tubing, two inches long. A rubber toy balloon is tied to each cork. In fitting the balloons to the corks it is best to pull the necks all the way up over the corks, thus preventing any possibility of their flying off when inflated. Care should be taken to have the balloons of equal distensibility. A number should be purchased in order to pick out two that are mates and they should be stretched by hand as equally as possible before first inflating them. In order to avoid changing the bronchial stenosis valves at each demonstration, it is well to have four corks fitted with glass tubing. One pair is left open; a sheet rubber flap is tied over the outer end of the third tube (check valve) and a similar rubber flap is tied over the inner end of the fourth (stop valve). The corks are easily changed and delay in changing and adjusting the valves during the demonstration is avoided.

The pump consists simply of two parallel cylinders of 3 inches diameter and about 14 inches effective stroke. The piston disks are fitted with leather washers pointing in both directions of the stroke. The piston rods are secured to a common handle. Hose nipples of ⅜ inch are furnished at the bottom ends of the cylinders. A hose runs from each cylinder to each chamber. There must be no interchange of air between the chambers, hoses, or pumps.

Editorials

The Proper Place of Transurethral Resection in Surgery of the Prostate

FOR some occult reason, the treatment of the obstructing prostate has always evoked bitter disputes. As early as 1850 there were arguments as to the propriety of sudden as compared to gradual emptying of the bladder. Toward the end of that century, castration had its advocates and opponents. A few years later the suprapubic operators were at swords' points with the perineal prostatectomists. By the time the writer became interested in urology, it was quite generally agreed that, while the mortality of suprapubic prostatectomy left something to be desired, at least it did not often cause urinary incontinence; the perineal operation had a lower mortality, but it was alleged by its opponents that the difference in mortality, while it favored the perineal approach, was represented by patients who, being incontinent with or without rectourethral fistulas, wished that they had died.

The other features of prostatic surgery were standardized: one must empty the distended bladder gradually; all patients must be "drained" before operation for at least ten days; the closure of the postoperative fistula, while usually tedious and accompanied by those fearful odors which set the "genitourinary ward" apart, was a necessary evil; and it was remarkable that such old men could be operated upon with any success at all.

Many surgeons realized that the obstructing prostate had been attacked through the intact urethra many years before but that such attempts had come to a bad end, except in a few patients with contracted prostates which could be benefited by operation with a misshapen cystoscope called Young's punch. True, Caulk had long proclaimed that, using an instrument resembling the Young punch, he could relieve retention due to prostatic hypertrophy as readily as that due to contraction, with a very low mortality, but no one paid much attention. Then all at once Davis reported similar results with the modified diathermy resectoscope of Stern; Bumpus found that he could do the same with the Braasch punch, and Alcock achieved remarkable results with McCarthy's instrument.

The battle was at once joined, with the advocates of perineal and suprapubic prostatectomy, their ancient quarrel temporarily forgot-

ten, on one side, and the resectionists, quarrelling a little among themselves over the relative merits of the diathermy loop and the cold punch, on the other.

At the present time one may say with considerable assurance that the average surgeon operates upon the prostate by the method which he learned first and likes best, and he is inclined to make the patient fit the operation; if he is especially interested in prostatic surgery, he is likely to defend his own methods and to condemn those of others with more heat than logic. Nevertheless, it is apparent to any well-informed and unprejudiced observer that fairly precise indications exist for the use of each of the three major surgical approaches to the prostate gland.

In deciding what these indications are, one must consider both the surgical and the pathologic factors involved. The objective of any operation upon the obstructing prostate is clearly to relieve completely the obstruction to urination with the least possible danger to the patient's life and to his control of urination, at the same time giving due weight to such factors as the avoidance of recurrence, the length of disability with its attendant expense for hospitalization, and the degree of discomfort and inconvenience to the patient.

How best to attain these objectives is necessarily influenced by the local conditions obtaining in the prostate. It is trite to say that benign hypertrophy is encapsulated and can be readily enucleated; that fibrosis is diffuse and is best treated by cutting a channel through it. Cancer occupies a variable position; rarely it may be embedded in and concealed by a spheroid of hypertrophy, with which it can be completely enucleated; it is then diagnosed only by the pathologist; just as rarely it may be completely confined within an otherwise normal gland which then invites total excision; most often it has extended locally or metastasized and thus requires palliative partial excision.

What are the possibilities of the three approaches in these conditions?

The suprapubic approach is easiest to master, requires no extensive armamentarium, and involves very little risk to the external sphincter. Its mortality averages 8 per cent in large series in good hands and is higher in average hands. Two weeks are usually consumed in preliminary drainage and four more, especially annoying and tedious to the patient and his attendants, in closing the fistula. It is thus uncomfortable and expensive for the patient. Moreover, it is really practicable only for the enucleation of benign hypertrophy, with or without a concealed cancer. The difficulty of anastomosing the bladder to the urethra from above practically precludes its use for total extirpation of the gland, and it is much inferior to the transurethral route for partial excision.

The perineal approach is harder to learn and much more exciting to perform because of the inviting proximity of the rectum and external vesical sphincter. While there are no accurate data as to the incidence of rectourethral fistulas and urinary incontinence, it is to be noted that some of the leading advocates of perineal prostatectomy have published detailed operations, complete with case reports, for the correction of these conditions. Nevertheless, the mortality in expert hands is a good 5 per cent lower than that of the suprapubic approach. The time consumed in preparation and in convalescence is approximately the same in each, and both operations are prone to leave mental scars upon the patient who has experienced the rigors of the malodorous wet period or the prolonged use of the inlying catheter by which this may be avoided.

The outstanding advantages conferred by the perineal approach are: the ability to excise, under direct vision, an area of suspicious induration; to subject it to fast frozen section diagnosis; and to remove the entire gland in localized cancer. Young's report of 50 per cent of five-year cures of cancer with this method is far and away the most cheerful aspect of prostatic cancer and justifies the considerable risk of incontinence, especially if one forgets that local extension or metastasis will limit its application to about 10 per cent of cancers of the prostate, thus cutting the cures from 50 per cent of those operated upon to 5 per cent of all prostatic cancers, a figure no better than that obtained with irradiation without risk to continence. It must be pointed out, however, that the patient who has had a radical perineal prostatectomy for cancer rarely suffers from recurrent obstruction even though he dies later from carcinomatosis. There is no doubt that this operation deserves more widespread consideration than it now receives.

Transurethral resection has its disadvantages as well as its advantages. One of its pitfalls is its apparent simplicity which, coupled with the high pressure salesmanship of some of the equipment manufacturers, has led many a general practitioner to venture into a field for which he is at least as ill trained as for brain surgery, which he would not dream of attempting. The fact is that the operation is a fussy one, requiring a great cystoscopic experience, considerable patience, and much practice. Like golf and swimming, it is not to be learned by watching others; unfortunately there are very few centers at which it may be learned by doing; and these centers quite properly train only their own residents. The practitioner who pays a fee for a three- to six-week course in transurethral resection is buying a dead horse, and the odor of its decay will sooner or later assail the nostrils of his colleagues, his patients, and their friends and relatives. Yet, it is done!

In expert hands and in favorable circumstances the mortality of transurethral resection may be less than 1 per cent (Caulk, Davis, Thompson), but it probably averages 3 or 4 per cent. The risk to control is minimal, notwithstanding the great increase in the sale of incontinence clamps which is said to have accompanied its spread. The writer has had one case of permanent incontinence in some 800 patients (0.125 per cent).

Alert, expert nursing care is essential during the first days of convalescence and is not available in many hospitals. The period of hospitalization is greatly reduced, Thompson having eliminated preliminary drainage in 60 per cent and reduced the postoperative stay in the average case to five days. While the total hospital stay will average ten to fourteen days in the less favorable conditions obtaining with indigent patients, this is an important gain over the forty-odd days required for prostatectomy, and is especially gratifying to the patient who undertakes to pay his own hospital bill. When one considers that the "wet period" during closure of the fistula is eliminated, one understands why a demand for this type of operation has developed among informed patients. What a pity that this demand has led untrained practitioners to attempt the operation "because the patient will go to someone else if he doesn't!"

It has been alleged that many, if not most, of the patients have dysuria, persistent frequency, and pyuria after resection. This is due to incomplete operation and appears with diminishing frequency as an operator becomes competent.

The transurethral operation is ideal for the relief of obstruction from fibrosis or from cancer which has extended locally or metastasized, not only because of the smaller risk, shorter hospitalization, and lessened discomfort as compared to prostatectomy, but also because partial removal of the gland is accomplished more readily and accurately than by open operation. The chief objection to transurethral resection arises in connection with benign hypertrophy, where it necessarily leaves abnormal tissue behind except in the relatively infrequent cases of pure posterior commissural or subtrigonal hypertrophy. Caulk believes that the abnormal tissue remaining after operation will atrophy if obstruction has been completely relieved, but this is hard to believe because it amounts to saying that the obstruction to urination caused the hypertrophy in the first place, when the reverse is obviously true.

No doubt recurrence will be common in any operator's first cases of hypertrophy; the question of its frequency in the hands of an experienced resectionist cannot be settled until many cases of benign hypertrophy have been followed for five or more years after adequate operation by competent operators. In the meantime, the risk of recur-

rence seems sufficiently remote to justify the continued application of the method to benign hypertrophy.

The precise indications for the three types of operation may best be summed up as follows: Transurethral resection is clearly indicated in prostatic fibrosis, in inoperable (in the sense of cure) obstructing cancer, and in benign hypertrophy of small to moderate size, and in most poor risks because of the lower mortality.

The moderately large hypertrophies (requiring the removal of 75 gm. or more) form the group in which the indications as to type of operation are not yet clearly defined. Here the transurethral operation may be so prolonged as to introduce an element of risk not inherent in the case of the smaller glands; operation may have to be done in two, or even three, stages, so that the advantage of shorter hospitalization is to some extent offset. The possibility of recurrence is greater, so that there is definite ground for maintaining that suprapubic or perineal prostatectomy is preferable.

In the large hypertrophies, requiring the removal of more than 100 gm. of tissue, suprapubic prostatectomy is the method of choice except for those surgeons who are well trained in perineal surgery. For localized cancer, radical perineal prostatectomy is unique in offering a fair prospect of cure.

To the surgeon who argues that he is better equipped personally to use one method where another seems clearly indicated, it is obvious that the patient rightfully belongs to the surgeon who is equipped to fit the operation to him, rather than to the man who trims the patient to fit the operation at his command.

—*C. D. Creevy, M.D.*
Minneapolis, Minn.

Abdominal Wall Susceptibility to Infection

FOR a considerable time there has been a great deal of controversy concerning the advisability of draining the peritoneal cavity in cases with peritoneal contamination and infection. Whereas Treves originally emphasized the importance of draining whenever in doubt, there has been recently a tendency toward the omission of drainage in those cases in which there is no localized suppurative process. This has been due largely to the fact that it is impossible to drain the peritoneal cavity per se for longer than a period of a few hours. The original, fundamental, investigative work of Yates demonstrated this beyond a doubt, and this has been subsequently corroborated by many clinical observations. Unquestionably, the introduction of a drain into the peritoneal cavity in cases of diffuse contamination or infection not only does little or no good except for the first few hours when the peritoneal cavity might be drained, but also probably it actually does harm by virtue of the presence of the foreign body. The danger of walling off of portions

of the peritoneal cavity with the production of residual abscesses is even greater when drains are used than when the peritoneum is closed without drainage.

The reason for the abolition of drainage of the peritoneal cavity is the fact that not only does the drain become walled off within a period of a few hours and because of this is unable to drain the peritoneal cavity per se, but also that the peritoneal cavity is able to withstand considerable infection. Inherent resistance of the peritoneal cavity to infection is well known.

Whereas the peritoneal cavity possesses considerable resistance to infection, the abdominal wall does not share in this resistance; in fact, it is particularly susceptible to bacterial contamination. Unfortunately, because of the trend recently of omitting drainage in contaminations and infections of the peritoneal cavity, this has been taken too literally, and many surgeons omit drainage of the susceptible abdominal wall. Every surgeon has had the experience of closing tightly the peritoneum, which has been contaminated either preoperatively or at the time of operation, and has, following closure of the abdominal wall, observed the development of an abdominal wall abscess. Although in the majority of instances this may be of no consequence, because, when the abscess does develop, it can be evacuated to the outside, in some instances the development of a phlegmon of the abdominal wall is dangerous. Whereas in any operation upon an intraabdominal viscus containing micro-organisms, there is potential contamination, the degree of contamination can be decreased materially by the employment of technical measures, such as aseptic intestinal suture, which minimizes contamination. In such cases contamination of the highly susceptible abdominal wall can be further decreased or eliminated by walling off the abdominal wall from the site of contamination by placing moist cloths not only around the external incision but also into the peritoneal cavity. Whereas emphasis of this technical detail may appear unnecessary, it is surprising how frequently the measure is overlooked.

If the abscess develops between the muscles and the peritoneum, its presence may not be detected and adequate external evacuation of its contents may not be accomplished. In such an instance spontaneous rupture of the abscess into the peritoneal cavity may occur. The author has observed such a case which terminated fatally within a period of eight hours after the rupture, due probably to the rapid absorption of toxic material from the peritoneal cavity in which the absorption was normal. Not infrequently, infection of the abdominal wall complicating a ruptured appendix is due to anaerobic organisms and is likely to progress unless aerobiosis is obtained. This has been emphasized recently by Gamble, who has materially reduced the mortality rate in the treatment of ruptured appendicitis by leaving the abdominal wound wide open.

Although one may feel that the extreme of not closing the abdominal wound in contaminated or infected wounds is too radical, it cannot be denied that in such cases the institution of drainage of the abdominal wall is desirable and often a life-saving procedure. In cases in which, during the operative procedure, contamination does occur, a strip of rubber tissue should be inserted into the wound lying just external to the peritoneal suture, which should be closed tightly. Over this the muscles and fascia are sutured and another strip of rubber dam is placed external to the muscle sheath. In this way any possible infection of the extraperitoneal and extrafascial spaces is drained. Generally it is not necessary to leave the drainage in for more than forty-eight hours, because by this time, unless suppuration has occurred, a sufficient sinus has developed to allow the evacuation of any material which may subsequently form.

—Alton Ochsner, M.D.

Recent Advances in Surgery

CONDUCTED BY ALFRED BLALOCK, M.D.

RECENT ADVANCES IN THE SURGICAL TREATMENT OF LESIONS OF THE BILIARY TRACT

WALTMAN WALTERS, M.D., D.SC., ROCHESTER, MINN.
(From the Division of Surgery, the Mayo Clinic)

IN AN attempt to evaluate the advances that have been made in the surgical treatment of lesions of the gall bladder and bile ducts in the past year, one cannot help but be impressed by the number of worthwhile contributions which have appeared in the literature as well as the character of the studies undertaken. To present these numerous advances in a paper of reasonable size of necessity means the elimination of reference to many excellent papers, and to some this may seem to have been an oversight. Because of limitation of space, however, reference will be made only to the more important contributions.

ANATOMIC STUDIES

Under this heading I have grouped not only anatomic studies of the cystic duct and sphincter of Oddi but also studies of the anatomic and physiologic features of the gall bladder and bile ducts by cholangiography or choledochography (roentgenographic studies after the injection of an opaque substance, such as brominol, lipiodol or hippuran, into the biliary tract).

There has been a good deal of speculation as to the development and function of the valves of Heister in the cystic duct. Following a series of experiments on human gall bladders within twelve to twenty-four hours after death, Lichtenstein and Ivy concluded that "the valves of Heister are an embryological formation caused by the winding or longitudinal compression of the duct during its development. The variations in the number and size of the 'valves' are due to the variations in the size and parts that take part in the foldings and windings, and the pressure variations on either side of the valvular portion of the cystic duct were not influenced by the presence of these valves."

Boyden's interesting anatomic studies on the sphincter of Oddi in man have been of practical assistance in evaluating the effect of spasm of the sphincter on the production of pain resembling gall-bladder colic, an intensive study of which has been carried out by McGowan, Butsch,

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and me,[30, 31, 45] and by Doubilet and Colp. These studies have shown that morphine, codeine, dilaudid, and pantopon, in the usual therapeutic dosage, frequently will produce spasm of the sphincter of Oddi, thus increasing the intraductal biliary pressure and producing pain. McGowan, Butsch, Knepper, and I[10, 31, 32] have demonstrated roentgenographically and by studies of intraductal pressure that the antispasmodics, amyl nitrite and nitroglycerin, will relax spasm of the sphincter, decrease intraductal pressure, and relieve pain. Recent work by McGowan and Knepper[32] in studying intraduodenal pressure in similar cases has shown that the same drugs have the same effect on the wall of the duodenum; in other words, coincident to spasm of the sphincter and an increase in intraductal pressure, spasm of the duodenum also occurs and relaxation takes place following the administration of amyl nitrite or nitroglycerin. Boyden has stated that: "the experimental work of the last twenty-five years indicates the existence of an intrinsic musculature surrounding the lower end of the common duct which, under morbid conditions, can produce biliary stasis. . . . The human sphincter has three marked anatomical characteristics: (1) its relative freedom from intestinal interference due to the configuration of the window in the duodenal muscle through which it passes, (2) the retrogression of its ampullary segment, and (3) the development of a special constricting mechanism (the sphincter choledochus) just above the site where the bile duct joins the ampulla of Vater." Further studies of the configuration of the gall bladder and bile ducts, both at the time of operation and subsequently, have been made by several investigators, among them being Mirizzi, who was one of the pioneers with Overholt, Bartlett, and others in this field of investigation. The papers by Mirizzi, Best and Hicken, and by Baker and Bacon on operative cholangiography are worthy of thorough study. Best and Hicken, instead of using oily substances like lipiodol or brominol, have found hippuran more satisfactory. Such studies will reveal the presence of stones in the common duct as well as causes of persisting ductal obstruction subsequent to operation and, in cases in which tubes have been left in the gall bladder or common duct for drainage, cholangiography has become almost a recognized routine procedure.

ETIOLOGY OF FORMATION OF GALLSTONES

Sir James Walton, in a most interesting paper on the formation and treatment of calculi in the biliary tract, called attention to the fact that, in his experience, the single cholesterin stone in the gall bladder has always been associated with chronic inflammation in the wall of the gall bladder: "if bile does not freely enter the gall bladder," he said, "a stone of pure cholesterin results. If it does, a mixed or laminated stone will result." Mackey, after studying 400 gall bladders removed by various surgeons, found that 81 showed cholesterosis; he concluded

that: "cholesterosis is not a manifestation of cholecystitis . . . and that cholesterosis and lithiasis are cognate manifestations of supersaturation of the bile with cholesterol. . . . Hence, the cause of characteristic cholesterin stones is primarily metabolic and not infective although inflammatory complications of mechanical origin are likely to supervene later."

Mackey's last point, I think, is worthy of emphasis, for the reason that removal of a gall bladder containing a single cholesterin stone seems to me to lie in the possibility of its obstructing the cystic duct, with the formation of an acute hydrops or empyema. Such impacted stones can equally well perforate the gall bladder, as stones of the mixed type do, and likewise can pass through the cystic duct into the common duct and lead to symptoms of obstruction. In a case in which I recently performed operation, a solitary cholesterin stone, about 1.5 cm. in diameter and producing symptoms of biliary obstruction, was removed from the ampulla of Vater.

Walton's comment on the increased frequency of stones in women being due to the dissemination of a *Bacillus coli* infection raises an interesting point in etiology, since in his experience 70 per cent of the patients who were operated on at the London Hospital had colon bacilli in their urine. The reason he considered pregnancy as a disposing factor in the formation of gallstone was because of its tendency to increase the incidence of *Bacillus coli* infections. In an interesting paper on various theories of gallstone formation, Illingworth credited Aschoff and Baemeister with having originated the present-day conception of gallstone formation. According to this idea, the variation in the appearance, structure, and mechanical composition of gallstones is clear proof of their different modes of origin, the four main types being: (1) single stones of pure cholesterol, (2) multiple, pure cholesterol stones, (3) bile-pigment stones (bilirubin and calcium), and (4) mixed stones (cholesterol, bilirubin and calcium).

Reflux of pancreatic juice into the gall bladder convinced Wolfer that in selected cases it was a cause of acute necrosis and acute gangrenous cholecystitis and that it might also, in some cases, be a cause of chronic cholecystitis with or without stones. Rufanov collected approximately 57 cases of formation of stones within the intrahepatic ducts that had been reported in the literature and reported 5 of his own. The frequency with which these stones form makes them of practical interest because this may explain the presence of stones in some cases in which the common duct has previously been operated upon and stones removed.

In discussing the etiologic factors in relation to hepatic lesions, Fitz-Hugh reported 7 cases of acute gonococcal perihepatitis which presented a syndrome suggesting acute cholecystic disease; in all but 1 of these cases urethral and cervical smears were positive for gonococci. The infection began to subside spontaneously in from one to two weeks and

the outlook was good although it was possible that the infection might recur. Coller and Forsbeck reported 18 cases in which cholecystectomy was performed on typhoid carriers. Cures were obtained in 88.9 per cent of the cases; all of the patients recovered from the operation.

ACUTE CHOLECYSTITIS

The debate on the question of early versus delayed operation in cases of acute cholecystitis continues. Walton, for example, has said: "In my own experience the risk of immediate operation has been no greater than waiting for weeks. Immediate operation should always be advocated." Bergh, on the other hand, in a most interesting paper on the treatment of acute cholecystitis in the surgical clinics of the University of Minnesota, said: "During the past few years a number of surgeons have advocated early operation in cases of acute cholecystitis. Many others, however, have condemned such a procedure as being more dangerous than the conservative policy of allowing the acute process to subside before operative interference is undertaken. The problem cannot be settled at this time." Both types of treatment were reviewed by Bergh and the method of conservative treatment in use at the University of Minnesota Hospitals outlined. One can assume from these studies that, unless acute perforation has manifested itself, definite benefit is obtained by delaying operation until the patient's general condition can be improved by such measures as subsidence of fever, restoration of fluids, and emptying of the gastrointestinal tract of fluid. Heuer, however, by carrying out immediate operation in cases of acute cholecystitis, reduced the mortality rate in 153 cases to 3.2 per cent. He believed that this decrease in mortality rate was due, among other things, to the prevention of gangrene and perforation of the gall bladder (which course of events is much more frequent than formerly thought to be the case), which may occur while one is waiting for the inflammatory process to subside.

Wesson, Montgomery, and I, in August, 1937, reported 87 cases of acute cholecystitis encountered at the Mayo Clinic during the period of two and one-half years from January, 1934, to July, 1936. In 76 of these cases operation was performed; in the remaining 11 cases, for one reason or another, it was deferred. In these 76 cases the diagnosis of acute cholecystitis was made by the surgeon at the time of operation or by the pathologist. There were 3 deaths, or a mortality rate of 3.9 per cent. In 2 of the cases in which death occurred, the gall bladder had ruptured and localized abscesses had formed; in the third case the gall bladder was gangrenous. In 16 cases (21 per cent) the gall bladder had ruptured, with localization of the infection, and in none was generalized peritonitis found at the time of operation. In 18 cases (about 24 per cent) the gall bladder was reported to be gangrenous. Cholecystectomy was performed in 61 cases (80 per cent); whereas, in the remaining

15 cases (20 per cent) the amount of acute inflammation present prevented proper exposure of the cystic and common ducts and cholecystostomy was performed. The general impression obtained from this review was that earlier operation would frequently eliminate perforation of the gall bladder and some postoperative complications, thus reducing the mortality rate.

Fiessinger, Bergeret, and Leveuf described hemorrhagic lesions of the gall bladder occurring without evidence of cholecystic infection. Such a condition was due to rupture of either the superficial capillaries or the deeper arterial vessels in the gall bladder, which, they believed, may have occurred as a result of stasis or irritation. Clinically, such hemorrhagic lesions of the gall bladder resembled those of acute cholecystitis, with unusually severe symptoms of pain and signs of peritoneal reaction. On palpation the gall bladder was found to be unusually hard.

Kalk stated that active inflammatory processes are contraindications to the use of the laparoscope, which method, however, he said, made it possible to determine the presence of carcinoma, cirrhosis of the liver, and gallstones. I am skeptical about the last, however, because, when stones are small and few in number, it is sometimes even difficult to feel them between the palpating fingers during abdominal exploration.

THE HEPATORENAL SYNDROME

Several interesting articles have dealt with the problem of death subsequent to operations on the biliary passages in cases in which the so-called hepatorenal syndrome is present. This syndrome has been described by Heyd as being responsible for some of the early deaths following operations on the biliary tract. The syndrome is characterized by an increase in temperature and pulse rate and by a progressive uremia and, finally, death.

In a study of the pathologic process in the liver and kidneys in such cases, Garlock and Klein emphasized the rôle of the kidneys in the production of the syndrome and found an initial stage of glomerular injury continuing on to the chronic form known to the clinician as "glomerulonephritis." The changes found in the liver were variable and rather insignificant when compared to those in the kidneys. Boyce and McFetridge, by sudden decompression of the biliary tract through the establishment of external biliary fistulas in experimental animals after first obstructing the biliary tract, were able to cause the death of the animals. Such animals died in three or four days and presented a clinical picture not dissimilar from that associated with the hepatorenal syndrome, and one characterized clinically by listlessness, anorexia, and anuria.

Repeating the work of Mann and Mason, Trusler and Martin found that dogs receiving intraperitoneal doses of from 30 to 100 gm. of liver usually died within twenty-four hours and showed all signs (including

the chemical changes in the blood) of shock. In addition, however, they found that peritoneal smears and cultures before death revealed anaerobic organisms. The parenchymal elements were found to be relatively nontoxic. However, 50 gm. of a suspension of liver connective tissue caused both shock and death from bacterial peritonitis. When the hepatic parenchyma alone was injected, the dogs survived and appeared well.

In further studies on surgical shock Harkins, Harmon, and Hudson attributed the toxic action of bile within the peritoneal cavity not only to the effect of the anaerobic bacteria but also to the escape of large quantities of the plasma-like fluid into the peritoneal cavity, which resulted in a concentration of blood, a fall in blood pressure, and a decrease in blood volume. The resulting shock, sufficiently reducing the resistance, makes the animal an easy victim to bacterial and toxic factors which would be less harmful to normal animals.

CAUSES OF DEATH IN DISEASE OF THE BILIARY TRACT

In studying the cause of death of 130 patients who came to necropsy, Colp and Ginzburg found suppurative cholangeitis ending in multiple hepatic abscesses to have been responsible for the death of 16 patients. In these cases the condition had progressed to the extent described as the result of years of operative delay. The effect of preoperative preparation and appropriate treatment of patients with obstructive jaundice who were operated upon was evidenced by the fact that but 3 deaths resulted from hemorrhage in their series of cases of obstructive jaundice. In another group of cases the condition was of the so-called interval type, in which group were placed patients whose disease at the time of operation was not threatening to life. Failure to recover in these cases they ascribed to errors in judgment or technique or to complications following operations (which at present seem to be unavoidable), such as pneumonia, which caused 11 per cent of the total deaths. Three deaths were caused by heart failure and 2 by pulmonary embolism. There were no cases in the group of so-called liver shock.

An interesting paper on the complications of gall-bladder surgery by Heyd contained an analysis of 557 of his personal cases; in 417 of these cases the patients were operated upon, with a mortality rate of 4.8 per cent. Gallstones were present in approximately 60 per cent of the cases in the series; whereas, 59 patients had ulcer of the stomach or duodenum associated with disease of the gall bladder. In 2 of the cases hyperpyrexia and coma followed surgical intervention and death ensued. Heyd concluded that the surgical treatment of disease of the gall bladder is safe and highly satisfactory. In 500 uncomplicated cases in which cholecystectomy was performed the mortality rate was 3.3 per cent.

In Finsterer's experience the most frequent lesion of the gall bladder is lithiasis and an accompanying cholecystitis. Although gallstone dis-

case often pursues a latent course, Finsterer nevertheless believed that any gall bladder containing stones should be removed. In his opinion, also, palliative operations relieving the obstruction were preferable to radical operations for carcinoma of the pancreas.

REVIEW OF SURGICAL RESULTS

Wilson, Lehman, and Goodwin reported a 3.6 per cent hospital mortality rate in 610 consecutive cases of cholecystitis and cholelithiasis. Studies in 447 of these cases by correspondence and re-examination showed that 91.3 per cent of the patients could be grouped as having obtained excellent, good, and fair results.

In reporting the results of operations for lesions of the gall bladder and biliary tract at the Mayo Clinic for the year 1936, Gray and I reported a total of 1,168 surgical procedures; 525 operations were performed for chronic cholecystitis alone, with 11 deaths, or a mortality rate of 2.1 per cent. In this group of cases jaundice was present in 46, or 8.8 per cent, and stones were found in the gall bladder in 88.8 per cent. When to this group were added those cases of acute cholecystitis in which patients were operated on as well as that additional group in which other major operations were performed in addition to the operations on the gall bladder, a total of 808 patients were found. In this series there were 26 deaths, or a mortality rate of 3.2 per cent. One hundred and forty-four (17.8 per cent) of these patients were jaundiced at the time of operation. The associated major surgical procedures consisted of exploration of the common duct in the majority of cases (78.8 per cent); whereas, operations were performed simultaneously on the gall bladder and gastrointestinal tract in 8.9 per cent of the cases in this group. Appendicectomy was not considered an additional major surgical procedure and, therefore, was not included.

Among 286 cases in which operation was performed at the same time for benign lesions of the gall bladder and bile ducts, there were 62 cases in which operation was on the common bile duct only, the gall bladder having previously been removed. In 130 of these 286 cases jaundice was present. The 62 patients who were operated upon for benign lesions of the bile ducts had, for the most part, stones, strictures, or fistulas of the common or hepatic ducts. Thirty-six of the 62 patients were jaundiced, and the operative mortality rate was 3.2 per cent.

I wish to stress particularly the frequency with which stones of the common duct are the cause of persisting symptoms of pain, sometimes bizarre in nature, or pylorospasm, frequently without typical biliary colics and even when a history of jaundice cannot be elicited. Many of the patients in this group had previously been denied operation elsewhere because of absence of a history of jaundice.

OBSTRUCTION OF THE COMMON BILE DUCT

Stricture.—A most excellent and interesting review of the reported cases of stricture of the common bile duct with the operation performed and the results of operation is included in a paper by Eliot published in the latter part of 1936. This is by far the most complete summary of such cases with which I am acquainted and very fittingly is a sequel to an article by the same author on a similar subject published twenty years ago. After a description of the various methods used and a discussion of the results to be expected, Eliot concluded as follows: "An accurate estimate of relative value of these operative procedures is impossible." That such a conclusion must of necessity be reached will be apparent to the readers of the article because of the fact that not only have the surgeons had to contend with strictures of varying extent, but, in addition, with livers showing a variable degree of damage and infection. The patients have passed into various stages of invalidism because of periods of incomplete, and often complete, biliary obstruction with infection.

My experience in 51 cases in which I performed operation for stricture (which cases were reported briefly in 1937[36, 46]) led me to believe that when the stricture is localized and there is sufficient duct remaining above it to allow accurate anastomosis to be made to the duodenum, this procedure, so-called choledochoduodenostomy or hepaticoduodenostomy, is followed by the best results. Even then, however, the results would be affected by the degree of infection within the liver and in the remaining portion of the duct above the stricture.

Lahey reported in detail 9 cases of stricture of the common duct. Each of these cases was chosen because treatment was carried out by a different plan and because the procedures had all been successful in overcoming the disease in a larger group of 35 cases in which operation was performed because of such complications. Following is a list of the procedures employed: (1) excision of the stricture with end-to-end anastomosis of the duodenum, a T-tube being brought out through a separate opening; (2) the Mikulicz procedure; (3) introduction of a rubber tube into the common duct with closure of the duct over the tube, the results of this being satisfactory for six and one-half months; (4) reconstruction over a T-tube in the case of narrow strictures; (5) choledochoduodenostomy; (6) end-to-end anastomosis of duct; (7) cholecystgastrostomy; (8) severance of constricting band; and (9) implantation of fistula into stomach and anastomosis of the stump of the cystic duct to the duodenum.

Hoag reported a case of stricture of the common bile duct in which an anastomosis was made between the common duct and the stomach, using the bell end of a catheter. After making a small opening in the stomach, the gastric mucous membrane was to be pulled up and sutured around the catheter. Bettman and Tannenbaum have modified the

Tate-Mason method of cholecystgastrostomy which consisted of making a small opening in the gall bladder by closing the opening with a purse-string suture around the bell end of a catheter. This purse string suture is kept long, and the catheter is passed through a small incision in the anterior wall of the stomach down into the duodenum. The ends of the purse-string suture are then threaded on a needle and passed through the lumen of the stomach and out the duodenum so as to serve as a tractor, thus telescoping the gall bladder into the incision in the stomach. These two structures are then held in this position with interrupted sutures.

Carcinoma.—In carcinoma of the head of the pancreas and in carcinoma of the ampulla of Vater the palliative operation of cholecystenterostomy or choledochoduodenostomy has long been the chosen method. Relief of jaundice and prolongation of life in such cases has been the experience of all surgeons. One of my patients lived for four and one-half years after cholecystgastrostomy. At post-mortem examination carcinoma of the head of the pancreas was found.

In a series of 113 cases in which tumors of the head of the pancreas were found to be the cause of the obstructive jaundice and in which cholecystenterostomy was performed at the Mayo Clinic, Dehne and I[47] found that 15 per cent of the patients were living and well five years or more after operation, which would lead one to assume that in these 15 per cent the process in the head of the pancreas had probably been inflammatory rather than malignant. The differential diagnosis can be made only by the removal of specimens for microscopic examination. However, the risk of such a procedure in the past has been thought to be too great, because bleeding is likely to occur from the cut area in the pancreas and leakage of pancreatic secretion into the free peritoneal cavity might occur, with serious consequences in either type of case, especially as an anastomosis between the biliary and gastrointestinal tract is to be made.

Oppenheimer reported the case of a patient who survived for twenty-nine months following cholecystgastrostomy for carcinoma of the head of the pancreas. The diagnosis was confirmed at necropsy. The absence of metastasis further shows the low degree of malignancy of carcinoma of the head of the pancreas, and the same is true of carcinoma of the ampulla. As a result, one must maintain an open mind relative to the feasibility of removing the first and second portions of the duodenum and a portion of the head of the pancreas for carcinoma of the ampulla or papilla of Vater and for carcinoma of the head of the pancreas, as advocated and carried out by Whipple, Parsons, and Mullins in 1935. Brunschwig performed such an operation, the patient surviving for approximately two and one-half months after removal of a large part of the duodenum and resection of the head of the pancreas, a preliminary anastomosis having been made between the biliary tract and the in-

testine and between the stomach and the intestine a month previously. At post-mortem examination, carcinoma of the peritoneum with ascites was found; there was metastasis throughout the liver and a duodenal fistula was present. In a case which I reported in 1932,[44] in which I had resected a portion of the duodenum for carcinoma of the ampulla, pancreatic metastasis was found at exploration a year later. An excellent summary of 18 cases in which radical resection of the ampulla of Vater was performed can be found in Hunt and Budd's paper, which was published in 1935. It includes a discussion of 59 cases previously collected and reported by Cohen and Colp.

It has been maintained by experimental workers that, following cholecystenterostomy in animals, ascending infection occurred in a high percentage of cases, producing cholangeitis and even hepatic abscesses. Such, however, has been the experience in clinical surgery only in exceptional cases. In order to decrease this possibility of intrahepatic infection, it was suggested that, after anastomosing the gall bladder or common duct to the duodenum, gastroenterostomy be performed in order to reduce intraduodenal pressure, thus assisting in preventing the reflux of ingested food through the anastomosis up into the biliary tract. In recent experimental studies Sandblom, Bergh, and Ivy, repeating such operations on experimental animals, found that even after pyloric exclusion some of the ingested material was found to make its way back into the duodenal loop. They therefore concluded that cholecystenterostomy and cholecystgastrostomy are satisfactory operations from the clinical standpoint and that more complicated procedures, such as an associated gastroenterostomy, add to the operative risk and are without definite advantage.

Other Types of Obstruction.—Obstruction of the common bile duct from tuberculous adenopathy of the hilus of the liver has been reported by Tantini, a tuberculous lymph node at the juncture of the cystic, hepatic, and common ducts producing a painful biliary obstruction with distention of the gall bladder. Pain and jaundice were relieved by removal of the lymph node. An aberrant blood vessel and connective tissue adhesions were found to be the cause of obstructive jaundice in 2 cases reported by Patterson. Their removal was followed by subsidence of the jaundice.

OVERLOOKED STONES IN THE COMMON DUCT

Particular stress should be placed on the frequency with which stones in the common duct are the cause of persisting symptoms of pain. This pain, as has been said, is sometimes bizarre. Pylorospasm, frequently without typical biliary colic, also is present in cases in which a history of jaundice cannot be elicited. Many of these patients may have previously been denied operation because of the absence of a history of jaundice or inability to visualize a stone in the common duct roentgenographically.

It has been my experience[49, 50] that most stones in the common bile duct cannot be demonstrated roentgenographically unless a method is available which allows the injection of an opaque substance into the common duct. With the more frequent visualization of the common bile duct subsequent to operation, the presence of overlooked stones will often be demonstrated. In one case in which the anomalous position of a large hepatic artery prevented exploration of the intrahepatic duct, and in spite of dilating the ampulla and the sphincter with exploring scoops at the time a stone was removed from the ampulla, postoperative choledochography[50] revealed the presence of a circumscribed shadow in the lower portion of the common bile duct after brominol was injected into it. Closure of the T-tube was followed by pain, clinical evidence of the presence of an obstruction. By instilling for a few seconds daily a few cubic centimeters of ether into the common duct the stone was fragmented, which fragmentation was demonstrated by choledochograms. By diluting the ether with alcohol, the T-tube could be closed for a longer time, and, by dilating the sphincter with amyl nitrite, the stony fragments were expelled into the duodenum. This, too, was verified by choledochography and by the fact that closure of the T-tube and its later removal were not followed by a return of pain.

Best has suggested, in cases of such overlooked stones, a method for increasing the intraductal pressure by the use of dehydrocholic acid, instilling at the same time warm olive oil through a tube into the common duct in order to relax the sphincter of Oddi.

It has been the custom at the clinic for many years to open and explore all enlarged common bile ducts, regardless of whether the patient has a history of obstruction of the duct and regardless of whether a stone can be palpated in it. The reason for this is that whenever a stone is present in the duct the duct is always dilated, and, although other conditions, such as pancreatitis, may cause enlargement of the duct, it is necessary to exclude stones. The advantages gained by drainage of the common bile duct in the presence of obstruction also emphasize the value of such a procedure. It is interesting that of 812 cases[48] of benign lesions of the gall bladder and bile ducts, the common bile duct was opened and explored in 182 of the cases because of dilatation and stones were found in the common or hepatic ducts in 109 cases, or 13.4 per cent. To put it another way, in 60 per cent of cases in which there was sufficient obstruction of the common bile duct to enlarge it, stones were present in the duct; whereas, in the other 40 per cent the causes of obstruction were inflammatory owing to an associated pancreatitis, with and without abnormal function of the sphincter of Oddi. This high incidence of stone in the common bile duct illustrates the point which we have made on many occasions, namely, that in the absence of a history of biliary obstruction stones in the common bile duct are likely to be overlooked. About this same incidence of stones in the common bile duct occurs in

that group of cases in which previous operations have been performed on the biliary tract, usually with removal of the gallbladder and subsequent to which the patient, because of recurring attacks of biliary colics, chills, or fever, underwent exploration of the common bile duct.

REFERENCES

1. Baker, H. L., and Bacon, C. M.: Lipiodol Visualization of Bile Tracts in Lesions with Jaundice, Surg., Gynec. & Obst. **65**: 220-227, 1937.
2. Bartlett, Willard, Jr.: Renal Complications of Biliary Tract Infections, Surg., Gynec. & Obst. **56**: 1080-1095, 1933.
3. Bergh, G. S.: A Critical Review of the Treatment of Acute Cholecystitis Including a Tabulation of the Experiences of the Surgical Clinic at the University of Minnesota, Internat. Abst. Surg. **66**: 29-37, 1938.
4. Best, R. R.: Discussion, J. A. M. A. **109**: 1596, 1937.
5. Best, R. R., and Hicken, N. F.: Technique of Immediate Cholangiography, Surg., Gynec. & Obst. **65**: 217-219, 1937.
6. Bettman, R. B., and Tannenbaum, W. J.: A Modification of the Tate Mason Method of Cholecystogastrostomy or Cholecystoduodenostomy, Ann. Surg. **106**: 465-467, 1937.
7. Boyce, F. F., and McFetridge, Elizabeth M.: The So-Called "Liver Death": an Experimental Study of Changes in the Biliary Ducts Following Decompression of the Obstructed Biliary Tree, Arch. Surg. **32**: 1080-1086, 1936.
8. Boyden, E. A.: The Sphincter of Oddi in Man and Certain Representative Mammals, SURGERY **1**: 25-37, 1937.
9. Brunschwig, Alexander: Resection of Head of Pancreas and Duodenum for Carcinoma—Pancreatoduodenectomy, Surg., Gynec. & Obst. **65**: 681-684, 1937.
10. Butsch, W. L., McGowan, J. M., and Walters, Waltman: Clinical Studies on the Influence of Certain Drugs in Relation to Biliary Pain and to the Variations in Intrabiliary Pressure, Surg., Gynec. & Obst. **63**: 451-456, 1936.
11. Cohen, Ira, and Colp, Ralph: Quoted by Hunt, V. C., and Budd, J. W.[25]
12. Coller, F. A., and Forsbeck, F. C.: The Surgical Treatment of Chronic Biliary Typhoid Carriers, Ann. Surg. **105**: 791-799, 1937.
13. Colp, Ralph, and Ginzburg, Leon: Mortality in Surgical Diseases of the Biliary Tract: an Analysis of One Hundred and Thirty Autopsies, Ann. Surg. **105**: 9-32, 1937.
14. Doubilet, Henry, and Colp, Ralph: Resistance of Sphincter of Oddi in the Human, Surg., Gynec. & Obst. **64**: 622-633, 1937.
15. Eliot, Ellsworth, Jr.: The Repair and Reconstruction of the Hepatic and Common Bile Ducts, Surg., Gynec. & Obst. **26**: 81-102, 1918.
16. Eliot, Ellsworth, Jr.: Benign Cicatricial Strictures of the Bile Ducts, Ann. Surg. **104**: 668-701, 1936.
17. Fiessinger, N., Bergeret, A., and Leveuf, J.: Hemorrhagic Gall Bladders, Internat. Abst. Surg. **66**: 46-47, 1938.
18. Finsterer, H.: Surgical Indications in Diseases of the Gall Bladder and Pancreas, Internat. Abst. Surg. **66**: 47, 1938.
19. Fitz-Hugh, Thomas, Jr.: Acute Gonococcic Perihepatitis—a New Syndrome of Right Upper Quadrant Abdominal Pain in Young Women, Rev. Gastroenterol. **3**: 125-131, 1936.
20. Garlock, J. H., and Klein, S. H.: The So-Called Hepato-Renal Syndrome, Ann. Surg. **107**: 82-95, 1938.
21. Harkins, H. N., Harmon, P. H., and Hudson, Jeanne: Lethal Factors in Bile Peritonitis. I. "Surgical Shock," Arch. Surg. **33**: 576-608, 1936.
22. Heuer, G. J.: The Surgical Aspects of Acute Cholecystitis, Ann. Surg. **105**: 758-764, 1937.
23. Heyd, C. G.: Complications of Gallbladder Surgery, Ann. Surg. **105**: 1-8, 1937.
24. Hoag, C. L.: Reconstruction of the Bile Ducts: New Method of Anastomosis, Surg., Gynec. & Obst. **64**: 1051-1054, 1937.
25. Hunt, V. C., and Budd, J. W.: Transduodenal Resection of the Ampulla of Vater for Carcinoma of the Distal End of the Common Duct, Surg., Gynec. & Obst. **61**: 651-661, 1935.
26. Illingworth, C. F. W.: The Formation of Gallstones, Edinburgh M. J. **43**: 481-497, 1936.

27. Kalk, H.: The Significance of Laparoscopy in Diseases of the Liver and Bile Passages, Surg., Gynec. & Obst. **64**: 559-560, 1937.
28. Lahey, F. H.: Strictures of the Common and Hepatic Ducts, Ann. Surg. **105**: 765-790, 1937.
29. Lichtenstein, M. E., and Ivy, A. C.: The Function of the "Valves" of Heister, SURGERY **1**: 38-52, 1937.
30. McGowan, J. M., Butsch, W. L., and Walters, Waltman: Pressure in the Common Bile Duct of Man: Its Relation to Pain Following Cholecystectomy, J. A. M. A. **106**: 2227-2230, 1936.
31. McGowan, J. M., Butsch, W. L., and Walters, Waltman: The Use of Glyceryl Trinitrate (Nitroglycerin) for the Control of Pain Following Cholecystectomy, Ann. Surg. **104**: 1013-1018, 1936.
32. McGowan, J. M., and Knepper, P. A.: Unpublished data.
33. Mackey, W. A.: Cholesterosis of the Gall-Bladder: a Review, Supplemented by Personal Observations on 87 Cases, Brit. J. Surg. **24**: 570-594, 1937.
34. Mann, F. C., and Mason, E. C.: Quoted by Dvorak, H. J.: Liver Autolysis in the Peritoneal Cavity of the Dog, Proc. Soc. Exper. Biol. & Med. **29**: 431-434, 1932.
35. Mirizzi, P. L.: Operative Cholangiography, Surg., Gynec. & Obst. **65**: 702-710, 1937.
36. Nygaard, K. K., Shelden, C. H., and Walters, Waltman: Strictures of the Bile Ducts: Results in Fifty-One Operative Cases, Proc. Staff Meet. Mayo Clin. **12**: 25-30, 1937.
37. Oppenheimer, G. D.: Prolonged Survival Following Cholecystogastrostomy for Obstructive Jaundice Due to Carcinoma of the Head of the Pancreas, Ann. Surg. **106**: 461-465, 1937.
38. Overholt, R. H.: Biliary Tract Visualization with Radiopaque Oils, Surg., Gynec. & Obst. **52**: 92-97, 1931.
39. Patterson, R. H.: Jaundice Due to Obstruction by an Aberrant Vessel and Adhesions, Ann. Surg. **104**: 1109-1112, 1936.
40. Rufanov, I. G.: Liver Stones, Ann. Surg. **103**: 321-336, 580-594, 1936.
41. Sandblom, Philip, Bergh, G. S., and Ivy, A. C.: Cholecystoduodenostomy Combined with Pyloric Exclusion, Ann. Surg. **104**: 702-711, 1936.
42. Tantini, E.: Obstruction of the Common Duct from Tuberculous Adenopathy of the Hilus of the Liver, Internat. Abst. Surg. **65**: 114-115, 1937.
43. Trusler, H. M., and Martin, H. E.: The Cause of Death in Liver Peritonitis: Blood Chemistry Findings in Dogs Subjected to Intraperitoneal Implantation of Fresh, Ground, Adult Dog Liver, SURGERY **1**: 243-254, 1937.
44. Walters, Waltman: Successful Resection of the Ampulla of Vater, Including a Portion of the Duodenum with Choledochoduodenostomy for Carcinoma of Ampulla of Vater, Surg., Gynec. & Obst. **55**: 648-651, 1932.
45. Walters, Waltman: The Pathological Physiology of Stone in the Common Bile Duct: Clinical and Surgical Significance, Surg., Gynec. & Obst. **63**: 417-424, 1936.
46. Walters, Waltman: Discussion, Ann. Surg. **105**: 787-789, 1937.
47. Walters, Waltman, and Dehne, E. A.: Jaundice Caused by Pancreatic Lesions, Surg., Gynec. & Obst. **54**: 832-835, 1932.
48. Walters, Waltman, and Gray, H. K.: Annual Report for 1936 of Surgery of the Stomach, Duodenum, and Biliary Tract, Proc. Staff Meet. Mayo Clin. **12**: 561-565, 1937.
49. Walters, Waltman, McGowan, J. M., Butsch, W. L., and Knepper, P. A.: The Pathologic Physiology of the Common Bile Duct: Its Relation to Biliary Colic, J. A. M. A. **109**: 1591-1597, 1937.
50. Walters, Waltman, and Wesson, H. R.: Fragmentation and Expulsion of a Common Duct Stone into the Duodenum by Using Ether and Amyl Nitrite, Surg., Gynec. & Obst. **65**: 695-697, 1937.
51. Walton, Sir James: The Formation and Treatment of Calculi in the Biliary Ducts and Gall Bladder, Surg., Gynec. & Obst. **64**: 257-269, 1937.
52. Wesson, H. R., Montgomery, T. R., and Walters, Waltman: Acute Cholecystitis: Report of Eighty-Seven Cases, Proc. Staff Meet. Mayo Clin. **12**: 500-505, 1937.
53. Whipple, A. O., Parsons, W. B., and Mullins, C. R.: Treatment of Carcinoma of the Ampulla of Vater, Ann. Surg. **102**: 763-779, 1935.
54. Wilson, W. D., Lehman, E. P., and Goodwin, W. H.: Prognosis in Gallbladder Surgery, J. A. M. A. **106**: 2209-2213, 1936.
55. Wolfer, J. A.: Pancreatic Juice as a Factor in the Etiology of Gallbladder Disease, SURGERY **1**: 928-938, 1937.

Book Reviews

Les Méthodes Chirurgicales du Traitement de L'Angine de Poitrine. Evolution et Resultats. By Marcel Bérard. Paper. Pp. 389. Paris, 1937, Masson & Cie.

In his monograph on *Les méthodes chirurgicales du traitement de l'angine de poitrine,* Marcel Bérard has presented a really excellent review of the results of surgical treatment of angina pectoris. No one has done this with any such thoroughness since Fontaine's thesis in 1925. Bérard, who has just completed his hospital training in Lyon, has had an exceptional opportunity to study the evolution of this subject. The cardiologists in Lyon rank among the first in Europe and Professor Leriche, in whose clinic he served, has had an unusually wide experience with the surgery of the autonomic nervous system. In addition to this, Bérard has had an opportunity to observe the work of the physiologists and surgeons in this country and to make a critical comparison of patients treated by neurosurgery (White), total thyroidectomy (Cutler), and vascular grafts to the myocardium (Beck). The fact that the author is a relatively young man without personal operative experience in this type of surgery is perhaps a good thing, because it gives him a more impartial viewpoint than has been shown by other older surgeons, biased as they are apt to be by their favorite operation.

The book contains an extensive review of the literature with references to some 240 publications. It is divided into chapters taking up the anatomy and physiology of the cardiac nerves, the mechanism of the anginal attack, and the various surgical procedures which have been tried to relieve it in cases which remain intractable to medical therapy. The chapter on anatomy of the cardiac nerves is well done, but should contain a few well-executed drawings. The increasing effectiveness of neurosurgical procedures for the relief of angina pectoris is principally due to a more accurate knowledge of the cardiac nerves whose pathways are much too complex to describe clearly by word alone.

Bérard has divided the possible methods of surgical attack into four main categories: (1) interruption of coronary constrictor reflexes; (2) blocking the sensory nerves from the heart; (3) decreasing the work of the heart and reducing the myocardial demand for blood by total thyroidectomy; (4) attempts to develop a collateral supply of blood to the myocardium through muscle grafts from the chest wall and pericardial adhesions. The greater part of the book deals with a description of the underlying physiology and the statistical results of these different methods which Bérard studied with Leriche and during his extended visit to the clinics in this country, where the surgery of angina pectoris is being developed. His conclusions are interesting and his comparative statistics of particular value as they have not been assembled heretofore.

He states that stellate ganglionectomy has been performed twenty-seven times by Leriche and Fontaine through a cervical approach under local anesthesia. Complete relief has resulted in 26 per cent, the severity and frequency of the attacks have been reduced in 41 per cent; failures are reported in 26 per cent and the results are uncertain in 7 per cent. The French surgeons deserve great credit both for their surgical skill and for their careful selection of cases which has enabled them to carry out this operation on one or both sides in 27 patients without a death. In explaining the physiologic action of stellectomy, Bérard follows the ideas of his teacher. As is well known, Leriche believes that a thrombosed artery sends

out vasoconstrictor reflexes, that in the case of the coronaries the stellate ganglia are the chief centers whereby this mechanism is set off, and that their resection should therefore abolish the anginal attack, not simply by interrupting the sensation of pain, but by increasing the circulation of the myocardium. If this hypothesis were correct, stellectomy would unquestionably be the ideal operation. But to those of us who have been indoctrinated by Cannon's ideas of homeostasis, it seems most unlikely that the sympathetic system, whose function is to prepare the organism for maximum exertion, should at the same time reduce the blood supply of the cardiac muscle. At the time this book went to press, the results of physiologic experiments were somewhat at variance as to which division of the autonomic nervous system carried the coronary constrictor and dilator fibers. However, Gollwitz-Meier and Krüger have recently measured blood flow through intact coronary arteries with the thermostromuhr of Rein and have demonstrated fairly conclusively that stimulation of the stellate ganglia causes coronary dilation rather than constriction. This confirms the earlier findings of Anrep and Segall and sets the weight of experimental evidence against the hypothesis upheld by Bérard. It indicates that stellate ganglionectomy acts through interruption of the principal sensory pathways from the heart, its failures being due to accessory afferent fibers in the direct thoracic cardiac nerves.

Bérard points out that stellectomy is unsuitable for patients with active decompensation or with evidence of advanced coronary changes in the electrocardiogram. For this group he recommends paravertebral alcohol injection of the upper thoracic sympathetic ganglia, as advocated by Swetlow and by White, or repeated novocainization as originally proposed by Mandl. The purpose of these injections is to block the cardiac pain fibers which reach the upper thoracic posterior roots over the sympathetic rami communicantes. The method is successful in direct ratio to the accuracy with which these delicate sympathetic rami have been blocked. After a personal study of the hospital records of 37 patients injected by White, he reports excellent results in 68.7 per cent and satisfactory improvement in another 17.6 per cent. He points out that the alcoholic neuritis of the intercostal nerves which follows injection may be troublesome and limits its use to the severest cases. Counter-balancing this objection, he feels that alcohol injection is undoubtedly the safest method and can be used after extensive coronary infarction and in other conditions which would make any form of surgery too hazardous.

Bérard also had the opportunity of seeing some of Cutler's patients after total thyroidectomy and of studying the records of 30 so treated at the Peter Bent Brigham Hospital. In this series, 10 per cent had complete relief and another 40 per cent considerable reduction in the frequency and severity of their anginal attacks. Another 40 per cent showed but minor improvement, and 10 per cent died. Bérard was much impressed by the troublesome myxedema which is a necessary corollary to this procedure. Furthermore, he points out that the myxedematous state may actually increase sclerosis in the coronary arteries by elevating blood cholesterol and the tendency to anginal attacks as a result of the accompanying anemia.

After observing Beck's cases treated by applying muscle grafts to the myocardium and inducing the formation of additional vascular adhesions to the pericardium, Bérard justly concludes that the results may be most impressive but that in its present state this operation carries too great a risk for its general adoption.

Bérard's book stands as a valuable contribution in a very specialized field and is highly recommended for those who have a reading knowledge of French and who are interested in the evolution of the much discussed problem of surgical intervention in the intractable forms of angina pectoris.

Legal Medicine and Toxicology. By Thomas A. Gonzales, Morgan Vance, and Milton Helpern. New York, N. Y., D. Appleton-Century Company.

The authors devote 448 pages to pathologic anatomy and 259 pages to toxicology. The legal phases are treated sketchily, but the anatomic problems are excellently illustrated and explained on the basis of the abundant experience of the authors. All types of injuries that a pathologist is likely to see are clearly discussed and explained. The chapter on bullet wounds is especially good. The chapter on sudden death is worthy of study by pathologists even if they are not interested in medico-legal problems.

It is correctly stressed that the medical examiner should examine the body where it is first found and take into consideration all the available evidence of whatever nature. He need not be an experienced detective, but he must be a shrewd and careful observer. The limitations of the pathologist are emphasized, and the discussions are well-balanced and sane.

In the discussions of death from poisoning, it is pointed out that the observations of the pathologist may be very helpful to the chemist in directing attention to the most probable form of poisoning. Full directions are given as to the organs and fluids which should be preserved for chemical examination.

The text is written in a clear, concise style and is amply illustrated. The book is the best available guide for pathologic anatomists engaged in medico-legal work.

Lehrbuch der Kriegs Chirurgie. Edited by Professors August Borchard, University of Charlottenberg, Berlin, and Victor Schmieden, University of Goethe, Frankfurt. Ed. 3. Pp. 821, with 464 illustrations. Leipzig, 1937. J. A. Barth. 34 RM.

It is now seventeen years since the second edition of this volume appeared under the title of *German Surgery in the World War*. Several familiar names are missing in the new edition. Death has taken Hermann Küttner, George Perthes, and Franz Rost—all well known to American students of surgery. Other senior authors have relinquished the responsibility of revision of their section to ambitious junior authors.

The subject matter embraces the entire scope of traumatic surgery in all its branches and includes discussion of the transport problem as well as that of after care. A new and worth-while addition is a chapter on roentgen technique; an equally serious omission is the discussion of Max Borst present in the second edition relating to the effects of gun-shot injuries on various tissues. The difficulty of differentiating the effects of Dum-Dum bullets, which are here described as of French origin (which they are not), from high velocity steel nosed bullets is admitted. The discussion of the treatment of shock suggests unfamiliarity with the concepts of Blalock and his associates with reference to its origin.

The chapter by Martin Kirschner on wound infection is particularly good. The physical, chemical, conservative, and operative treatments of infection are well discussed. The author, however, endorses Bier's hyperemia which, in the light of the studies of Landis on capillary filtration, would appear to be an unphysiologic procedure. Otto Goetze, of Erlangen, a new contributor, has an excellent chapter on injuries of the thorax and the section by Victor Schmieden of Frankfurt though containing little that is new, not contained in the second edition, is very informative.

There is of necessity some duplication of effort in a book such as this, which treats essentially of gun-shot injuries in which the resulting wounds are almost invariably complex and rarely involve single tissues. There is, however, much in-

formation in the volume of value to the civil surgeon, even though the aim of the book is to set forth the methods and results of treatment of war injuries.

The general format of the book is not so pleasing as that of the second edition which has a bolder and larger type with better reproduction of illustrations. The larger audience reached by a less expensive book containing the same subject matter well justifies these measures of economy. Ernst Herter's figure of the dying Achilles extracting the poisoned arrow from his heel adorns the frontis cover.

The Laboratory Diagnosis of Syphilis. By Harry Eagle. Pp. 440, with 27 illustrations. St. Louis, 1937, The C. V. Mosby Co. $5.

This extensive book represents one of the finest of recent contributions to the field of serology in syphilis. It includes a thorough study and a discussion of every problem encountered in this field, giving a detailed analysis, discussion, and description of every step in the Wassermann and flocculation tests with serum and spinal fluid; it includes theory, technique, and clinical interpretation.

This book is invaluable to serologists and syphilologists, and to general practitioners interested in this field. It should do much to bring a better understanding between the clinician and the serologist. It should help bring us a step closer to a standardized and simplified laboratory test to replace the various complicated Wassermann techniques.

The Postmortem Examination. By Sidney Farber, M.D. Pp. 201, with 32 illustrations. Springfield, Ill., 1937, Charles C. Thomas. $3.50.

This little book is a manual outlining autopsy technique. It gives a very short historic sketch, emphasizing the milestones in the development of the science of postmortem examination, with some interesting facts concerning its early history in this country. The author points out that the autopsy room is no longer the dead house, hidden away in some dark spot; but in the modern medical institution, it is comparable to a well-run surgical operating room in its appointments, cleanliness, and decorum. He goes on to outline in detail the various accepted techniques. The importance of a legal permission and the matter of cooperation with the undertaker is discussed. Useful tables giving normal organ weights at various ages are appended. The parathyroid weights are new and will be found particularly useful. The illustrations are, for the most part, drawings. They leave something to be desired as to number, but they show the points to be demonstrated fairly clearly.

As the author points out, autopsy technique is best learned in the autopsy room under the supervision of an experienced pathologist. No book can replace an apprenticeship, but the volume will be found useful for those who are responsible for the teaching of internes and graduate students. The publication of this and a similar book this year indicates a revival of interest in careful autopsy technique. The author's statement in the preface, to the effect that the book will make "available in the English language a description of methods used both here and abroad," neglects the fact that the works of Wadsworth and the earlier manual of Hektoen, while they may be out of print, are still available in most medical libraries. These books are still valuable in that the work of Wadsworth is more complete than the present volume. Changes in autopsy technique have been insufficient to render them obsolete.

Synopsis of Ano-Rectal Diseases. By Louis J. Hirschman. Pp. 288, with 174 illustrations, 6 in color. St. Louis, 1937, The C. V. Mosby Company. $3.50.

As the title implies, this edition is essentially a "condensed work" dealing with the diagnosis and treatment of the commoner diseases of the anus and rectum. The medical student and the general practitioner should be interested in this excellent summary.

The author includes a general discussion of the most commonly found conditions of the anus and rectum. There are also sections concerning the anatomy of the anal canal and rectum, the types of anesthesia employed, symptomatology associated with ano-rectal disease, and the technique of a carefully executed proctologic examination. Mention is made as to the necessary instruments that are to be included in a proctologic armamentarium.

Little consideration is given to the important subject, cancer of the anus and rectum. The use of setons and the injection of bismuth paste in the diagnosis and treatment of fistulous tracts is mentioned; "they should be tried only in those cases where operative procedures are refused or not thought advisable." The author takes into consideration the subject of anal fistula in the tuberculous patient and discusses this subject briefly but effectively. The section concerning proctitis and sigmoiditis, in which atrophic and hypertrophic proctitis are mentioned, is subject to comment. Very little mention is made as to the specific and nonspecific inflammatory lesion of the rectum and sigmoid. Rectal strictures are discussed, but very little emphasis is placed on this most important condition. Aside from the injection and cauterization treatment, there is no mention of other forms of therapy applied to prolapse of the rectum. A discussion of the congenital lesions of the anus and rectum is not included.

As in all compendiums, there are definite limitations and this one is no exception to the rule. It is well illustrated with drawings and photographs. The bibliography is quite brief. The author's personal remarks and suggestions gathered from a large private practice add considerably to the value of this volume. It is best suited for the medical student and the general practitioner, for whom it was especially prepared. For these two groups it should serve as an excellent summary.

Demonstrations of Physical Signs in Clinical Surgery. By Hamilton Bailey, Surgeon, Royal Northern Hospital, London. Cloth. Ed. 6. Pp. 284, with 358 illustrations, some in color. Baltimore, 1937, William Wood and Company. $6.50.

In all medical schools a course in physical diagnosis is given by the department of internal medicine. It does not propose to teach the physical signs of surgical diseases. Nor have teaching departments of surgery generally recognized the great importance of acquainting medical students early in their careers with the technique of examining surgical diseases. To be certain, every surgical department teaches the basic principles of surgery toward the close of the second or in the very beginning of the third year. This instruction, however, does not purport to orient the student in the technique of the examination of surgical disorders. It is time that teachers of surgery lend more serious thought to the great importance of instructing students of medicine in the physical signs of surgical diseases. How can the recognition of surgical disorders be learned by students unfamiliar with the methods of examinations?

It is with the techniques of examination that this splendid volume deals. It is eminently practical, concise, and well illustrated. Every medical student should have an acquaintance with it and would profit much by its study. It will prove of value,

also, to the general practitioner who desires ready access to a manual in which the critical differential points in physical examination of surgical disorders are reviewed.

There is much to commend in the volume and little to criticize. The inclusion of a few references would enhance the value of the volume to the student. The author has emphasized unduly the unphysiologic Loewi test of mydriasis with instillation of adrenalin in the eye in pancreatic disease. The initiation of visible peristaltic waves in acute intestinal obstruction by dropping ether on the abdominal wall is certainly demanding of more proof. The reviewer's experience leads him to believe that visible or palpable peristalsis, in the presence of an abdominal wall of normal thickness, is rarely, if ever, observed in acute obstructions. On the contrary, it is usually present in the obstruction which accompanies a stricture of the bowel. The counsel offered with reference to the recognition and management of paralytic ileus seems an unwarranted procedure that many American students of surgery would condemn, viz.: "Give the patient one (and only one) hypodermic injection of 1 c.c. pituitrin to stimulate peristalsis and wait for half an hour. If he vomits, operate; if nothing happens, operate; if his bowels are open, leave well alone." Most American surgeons have adopted as their credo and practice the passive treatment of physiologic paralytic ileus with constant suction applied to an inlying duodenal tube.

This volume has increased considerably in usefulness since the first appearance in 1927. It deserves to be widely read.

Practical Proctology. By Louis A. Buie. Cloth. Pp. 512, with 152 illustrations. Philadelphia, 1937, W. B. Saunders. $6.50.

This text by a practical man of big experience in his field will be welcomed by all who have an interest in diseases of the anal canal and rectum. The technique of examination, the best mode of management of cryptitis, hemorrhoids, anal pruritis fissure, and fistula, are well described.

The chief weakness of the text is that the discussion of some topics of the text is incomplete. The title suggests that diseases of the rectum and anal canal will constitute the subject matter. Yet such diseases as chronic ulcerative colitis, amebic dysentery, diverticuli and polyposis of the colon are well described at some length; whereas, the treatment of malignant disease of the rectum, which subject one might reasonably expect should be considered exhaustively, is by no means adequately dealt with. The techniques of the more formal operations which the surgeon performs most often for carcinoma of the rectum are not described. The choice of operative procedure in such conditions is not discussed. How imperforation of the rectum and anal canal should be managed surgically receives no mention. The operative relief of prolapse of the rectum is not discussed as comprehensively as the importance of the subject justifies. If chapters had been included detailing the broader viewpoint of the general surgeon with reference to these problems, the value of the text would have been enhanced considerably.

In view of the fact that the text does not concern itself alone with proctology but many diseases of the colon are discussed at length, a more appropriate title would appear to be proctologic aspects of diseases of the colon and rectum.

The text is accompanied by an excellent bibliography and a useful index. The author's ingenuous humor creeps out frequently. The opinions of Dr. Buie expressed in this monograph can be recommended for study by all who wish practical and authoritative information on common ills of the rectum.